ILE 'HOME' ENTERED AT 10:13:07 ON 29 MAY 2003

=> index bioscience medicine meetings
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...' ENTERED AT 10:13:33 ON 29 MAY 2003

79 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
search error messages that display as 0* with SET DETAIL OFF.

=> s diabetes and galectin? or ((macrophage (w) lectin (w) specific (s) galactose) and expression
UNMATCHED LEFT PARENTHESIS 'OR ((MACROPHAG'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s diabetes and (galectin? or (macrophage (w) lectin (w) specific (s) galactose)) and expression
```
         10   FILE BIOSIS
          1   FILE BIOTECHABS
          1   FILE BIOTECHDS
          1   FILE BIOTECHNO
          2   FILE CANCERLIT
         12   FILE CAPLUS
  15 FILES SEARCHED...
         10   FILE DGENE
          2   FILE DRUGU
  31 FILES SEARCHED...
          4   FILE EMBASE
          1   FILE ESBIOBASE
          0*  FILE FEDRIP
          1   FILE JICST-EPLUS
          1   FILE LIFESCI
          7   FILE MEDLINE
  48 FILES SEARCHED...
          2   FILE PASCAL
          1   FILE PROMT
          4   FILE SCISEARCH
          4   FILE TOXCENTER
        191   FILE USPATFULL
          5   FILE USPAT2
  64 FILES SEARCHED...
          1   FILE WPIDS
          1   FILE WPINDEX
          1   FILE NLDB
```

  22 FILES HAVE ONE OR MORE ANSWERS,   79 FILES SEARCHED IN STNINDEX

L1   QUE DIABETES AND (GALECTIN? OR (MACROPHAGE (W) LECTIN (W) SPECIFIC (S) GAL
       ACTOSE)) AND EXPRESSION

=> file hits
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	3.85	4.06

FILE 'USPATFULL' ENTERED AT 10:17:56 ON 29 MAY 2003

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FILE 'CANCERLIT' ENTERED AT 10:17:56 ON 29 MAY 2003

FILE 'DRUGU' ENTERED AT 10:17:56 ON 29 MAY 2003
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FILE 'WPINDEX' ACCESS NOT AUTHORIZED

FILE 'NLDB' ENTERED AT 10:17:56 ON 29 MAY 2003

```
=> s l1
L2          191 FILE USPATFULL
L3           12 FILE CAPLUS
L4           10 FILE BIOSIS
L5           10 FILE DGENE
L6            7 FILE MEDLINE
L7            5 FILE USPAT2
L8            4 FILE EMBASE
L9            4 FILE SCISEARCH
L10           4 FILE TOXCENTER
L11           2 FILE CANCERLIT
L12           2 FILE DRUGU
L13           2 FILE PASCAL
L14           1 FILE BIOTECHDS
L15           1 FILE BIOTECHNO
L16           1 FILE ESBIOBASE
L17           1 FILE JICST-EPLUS
L18           1 FILE LIFESCI
L19           1 FILE PROMT
L20           1 FILE WPIDS
L21           1 FILE NLDB

TOTAL FOR ALL FILES
L22         261 L1

=> s l22 and galectin-3
L23          13 FILE USPATFULL
L24           9 FILE CAPLUS
L25           8 FILE BIOSIS
L26           1 FILE DGENE
L27           5 FILE MEDLINE
L28           0 FILE USPAT2
L29           2 FILE EMBASE
L30           2 FILE SCISEARCH
L31           4 FILE TOXCENTER
L32           1 FILE CANCERLIT
L33           2 FILE DRUGU
L34           1 FILE PASCAL
L35           1 FILE BIOTECHDS
L36           1 FILE BIOTECHNO
L37           1 FILE ESBIOBASE
L38           0 FILE JICST-EPLUS
L39           1 FILE LIFESCI
L40           0 FILE PROMT
L41           0 FILE WPIDS
L42           0 FILE NLDB

TOTAL FOR ALL FILES
L43          52 L22 AND GALECTIN-3

=> dup rem l43
DUPLICATE IS NOT AVAILABLE IN 'DGENE'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L43
L44          31 DUP REM L43 (21 DUPLICATES REMOVED)

=> d l44 1-31 ibib abs

L44  ANSWER 1 OF 31  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER:    2003:182799  BIOSIS
DOCUMENT NUMBER:     PREV200300182799
TITLE:               Monocyte chemoattractant protein-1: Does it play a role in
                     diabetic nephropathy.
```

AUTHOR(S): Wada, Takashi (1); Yokoyama, Hitoshi; Matsushima, Kouji; Kobayashi, Ken-ichi
CORPORATE SOURCE: (1) Department of Gastroenterology and Nephrology, Graduate School of Medical Science and Division of Blood Purification, Kanazawa University, 13-1 Takara-machi, Kanazawa, 920-8641, Japan: twada@medf.m.kanazawa-u.ac.jp Japan
SOURCE: Nephrology Dialysis Transplantation, (March 2003, 2003) Vol. 18, No. 3, pp. 457-459. print. ISSN: 0931-0509.
DOCUMENT TYPE: Editorial
LANGUAGE: English

L44 ANSWER 2 OF 31 USPATFULL
ACCESSION NUMBER: 2003:93067 USPATFULL
TITLE: Reagents and methods for identifying and modulating **expression** of genes regulated by CDK inhibitors
INVENTOR(S): Poole, Jason, Chicago, IL, UNITED STATES
Chang, Bey-Dih, Lombard, IL, UNITED STATES
Roninson, Igor B., Wilmette, IL, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003064426	A1	20030403
APPLICATION INFO.:	US 2001-861925	A1	20010521 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-265840P	20010201 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE 3200, CHICAGO, IL, 60606
NUMBER OF CLAIMS: 94
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 3443
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides methods and reagents for identifying compounds that inhibit the induction of genes involved in cancer and age-related diseases, such genes being induced by cyclin-dependent kinase inhibitors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 3 OF 31 USPATFULL
ACCESSION NUMBER: 2003:57517 USPATFULL
TITLE: **Galectin** 9 and 10SV polynucleotides
INVENTOR(S): Ni, Jian, Rockville, MD, UNITED STATES
Gentz, Reiner L., Silver Spring, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PATENT ASSIGNEE(S): Human Genome Sciences, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003040081	A1	20030227
APPLICATION INFO.:	US 2002-235674	A1	20020906 (10)
RELATED APPLN. INFO.:	Division of Ser. No. US 2000-656450, filed on 6 Sep 2000, GRANTED, Pat. No. US 6468768 Continuation of Ser. No. US 1999-263689, filed on 5 Mar 1999, PENDING Division of Ser. No. US 1997-946914, filed on 9 Oct 1997, GRANTED, Pat. No. US 6027916		

	NUMBER	DATE

PRIORITY INFORMATION: US 1996-28093P 19961009 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: STERNE, KESSLER, GOLDSTEIN & FOX P.L.L.C., 1100 NEW YORK AVENUE, N.W., SUITE 600, WASHINGTON, DC, 20005-3934
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Page(s)
LINE COUNT: 2768
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel **galectin** 8, 9, 10 and 10SV proteins which are members of the **galectin** superfamily. In particular, isolated nucleic acid molecules are provided encoding the human **galectin** 8, 9, 10 and 10SV proteins. **Galectin** 8, 9, 10 and 10SV polypeptides are also provided as are vectors, host cells and recombinant methods for producing the same. The invention further relates to screening methods for identifying agonists and antagonists of **galectin** 8, 9, 10 or 10SV activity. Also provided are diagnostic and therapeutic methods.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 4 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
ACCESSION NUMBER: 2003:232619 BIOSIS
DOCUMENT NUMBER: PREV200300232619
TITLE: Protective effect of aminoguanidine on erectile function in **diabetes** induced rats.
AUTHOR(S): Usta, Mustafa F. (1); Bivalacqua, Trinity J. (1); Monnier, Vincent M.; Sell, David R.; Sanabria, Jose (1); Sikka, Suresh C. (1); Hellstrom, Wayne (1)
CORPORATE SOURCE: (1) New Orleans, LA, USA USA
SOURCE: Journal of Urology, (April 2003, 2003) Vol. 169, No. 4 Supplement, pp. 306. print.
Meeting Info.: 98th Annual Meeting of the American Urological Association (AUA) Chicago, IL, USA April 26-May 01, 2003 American Urological Association
. ISSN: 0022-5347.
DOCUMENT TYPE: Conference
LANGUAGE: English

L44 ANSWER 5 OF 31 DRUGU COPYRIGHT 2003 THOMSON DERWENT
ACCESSION NUMBER: 2003-11235 DRUGU P
TITLE: Managing heart disease. Mechanisms of cardiovascular complications in **diabetes** and potential new pharmacological therapies.
AUTHOR: He Z; Rask Madsen C; King G L
CORPORATE SOURCE: Joslin-Diabetes-Center
LOCATION: Boston, Mass., USA
SOURCE: Eur.Heart J. (5, Suppl. B, B51-57, 2003) 109 Ref.
CODEN: EHJODF ISSN: 0195-668X
AVAIL. OF DOC.: Joslin Diabetes Center, Section for Vascular Cell Biology, One Joslin Place, Room 4504, Boston, MA 02215, U.S.A. (G.L.K.).
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature
AN 2003-11235 DRUGU P
AB Management of heart disease and the mechanisms of cardiovascular complications in **diabetes** and potential new pharmacological therapies are reviewed. Insulin sensitive and insulin resistant cardiovascular mechanisms (mediators of vasomotion, nitric-oxide (NO) and endothelin-I, vascular endothelial growth factor (VEGF), a mediator of

angiogenesis, proliferation and apoptosis and cardiac substrate metabolism) are discussed. Mechanisms of hyperglycemia induced vascular damage (the advanced glycation end-product (AGE) theory, the reactive oxygen species theory, the protein kinase C (PKC) theory, and the cross-talk between these theories) are presented. Therapeutic strategies (targeting advanced glycation-end products, antioxidants, signaling molecules as targets, and anti-inflammatory agents) are all discussed.

ABEX A decrease in NO production in insulin resistance and **diabetes** is important in diabetic vascular complications. The insulin-stimulated vasodilatation is dependent on endothelium-derived NO. The VEGF regulates vascular permeability and angiogenesis, and can improve clinical outcomes in ischemic heart disease. Insulin has an antiapoptotic effect in endothelial cell culture. The myocardium utilizes fatty acids than glucose as an energy substrate. The improvements of insulin sensitivity of cardiovascular mechanisms prevent the diabetic complications. The AGEs can alter cellular functions by binding to the receptors for AGEs, (RAGE) or to the macrophage scavenger receptor, p60, p90 and **galectin-3**. The oxidative phosphorylation of glucose in mitochondria generates superoxide anion, the production of which increases with the hyperglycemia. The PKCs regulate vascular permeability, vasodilator release, endothelial activation, cardiomyocyte contractility and growth factor signaling. The over-**expression** of PKC-beta-2 results in a severe ventricular hypotrophy, interstitial fibrosis, cardiomyocyte necrosis and impaired contractility reminiscent of diabetic cardiomyopathy. Aminoguanidine prevent the complications of **diabetes** and the soluble range prevents the hyperglycemia. Antioxidants (vitamin E) block the microvascular complications of **diabetes**. LY-333531 can prevent or reverse early hemodynamic changes observed in diabetic retinopathy. The inactivation of IkappaB kinase can be prevented by high-dose aspirin and tumor necrosis factor-alpha, while infliximab improves endothelial dysfunction in rheumatoid arthritis. (SR/NK)

L44 ANSWER 6 OF 31 USPATFULL

ACCESSION NUMBER: 2002:272849 USPATFULL
TITLE: **GALECTIN** 8,9,10 AND 10SV
INVENTOR(S): NI, JIAN, ROCKVILLE, MD, UNITED STATES
GENTZ, REINER L., SILVER SPRING, MD, UNITED STATES
RUBEN, STEVEN M., OLNEY, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002150970	A1	20021017
APPLICATION INFO.:	US 1999-263689	A1	19990305 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 1997-946914, filed on 9 Oct 1997, PATENTED

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-28093P	19961009 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: STERNE KESSLER GOLDSTEIN & FOX, 1100 NEW YORK AVENUE N W, SUITE 600, WASHINGTON, DC, 200053934
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Page(s)
LINE COUNT: 2786
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel **galectin** 8, 9, 10 and 10SV proteins which are members of the **galectin** superfamily. In particular, isolated nucleic acid molecules are provided encoding the human **galectin** 8, 9, 10 and 10SV proteins. **Galectin** 8, 9, 10 and 10SV polypeptides are also provided as are vectors, host cells and recombinant methods for producing the same. The invention

further relates to screening methods for identifying agonists and antagonists of **galectin** 8, 9, 10 or 10SV activity. Also provided are diagnostic and therapeutic methods.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 7 OF 31 USPATFULL

ACCESSION NUMBER:	2002:272810 USPATFULL			
TITLE:	Bardet-biedl susceptibility gene and uses thereof			
INVENTOR(S):	Sheffield, Val, Iowa City, IA, UNITED STATES Nishimura, Darryl, Coralville, IA, UNITED STATES Stone, Edwin, Iowa City, IA, UNITED STATES			
PATENT ASSIGNEE(S):	The University of Iowa Research Foundation (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002150931	A1	20021017	
APPLICATION INFO.:	US 2001-25187	A1	20011218	(10)
	NUMBER	DATE		
PRIORITY INFORMATION:	US 2000-256900P	20001219 (60)		
	US 2000-258949P	20001229 (60)		
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	Steven L. Highlander, FULBRIGHT & JAWORSKI L.L.P., SUITE 2400, 600 CONGRESS AVENUE, AUSTIN, TX, 78701			
NUMBER OF CLAIMS:	67			
EXEMPLARY CLAIM:	1			
LINE COUNT:	5311			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to the identification of a gene, now designated negevin (ngvn), that is involved in the genetic disease Bardet Biedl Syndrome (BBS), which is characterized by such diverse symptoms as obesity, **diabetes**, hypertension, mental retardation, renal cancer and other abnormalities, retinopathy and hypogonadism. The human NGVN protein disclosed herein is 731 amino acids in length and is coded for by a gene spanning 17 exons. Homologs have been identified in mouse, rat, zebrafish. Methods of use for the gene, for example in diagnosis and therapy of BBS and in drug screening, also are described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 8 OF 31 USPATFULL

ACCESSION NUMBER:	2002:259402 USPATFULL			
TITLE:	IMMUNE ACTIVATION BY DOUBLE-STRANDED POLYNUCLEOTIDES			
INVENTOR(S):	KOHN, LEONARD D., BETHESDA, MD, UNITED STATES SUZUKI, KOICHI, NORTH BETHESDA, MD, UNITED STATES MORI, ATSUMI, BETHESDA, MD, UNITED STATES IISHI, KEN, ROCKVILLE, MD, UNITED STATES KLINMAN, DENNIS M., POTOMAC, MD, UNITED STATES RICE, JOHN M., WEST CHESTER, OH, UNITED STATES			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002142974	A1	20021003	
APPLICATION INFO.:	US 1998-151612	A1	19980911	(9)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	Steven J. Goldstein, FROST BROWN TODD LLC, 2200 PNC Center, 201 East Fifth Street, Cincinnati, OH, 45202			
NUMBER OF CLAIMS:	46			
EXEMPLARY CLAIM:	1			

NUMBER OF DRAWINGS: 22 Drawing Page(s)
LINE COUNT: 4436
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Double-stranded polynucleotide activates the **expression** of immune recognition molecules. The polynucleotide can have a minimal length and activates the **expression** of molecules not encoded by a nucleotide sequence that is not necessarily related to the polynucleotide. The present invention provides for a simple and specific system to activate **expression** of Class I and/or Class II molecules of the major histocompatibility complex (MHC), and allows regulation of **expression** of MHC molecules on the cell-surface of antigen presenting cells and other immune cells. Also provided are systems for the screening, identification, and isolation of compounds that increase or decrease this activation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 9 OF 31 USPATFULL
ACCESSION NUMBER: 2002:235500 USPATFULL
TITLE: Novel human **galectins**
INVENTOR(S): Hillman, Jennifer L., San Jose, CA, UNITED STATES
Goli, Surya K., Sunnyvale, CA, UNITED STATES
Bandman, Olga, Mountain View, CA, UNITED STATES
Hawkins, Phillip R., Mountain View, CA, UNITED STATES
Petithory, Joanne R., Fremont, CA, UNITED STATES
PATENT ASSIGNEE(S): Incyte Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002127689	A1	20020912
APPLICATION INFO.:	US 2001-894526	A1	20010627 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1998-206622, filed on 7 Dec 1998, ABANDONED Division of Ser. No. US 1997-788584, filed on 23 Jan 1997, GRANTED, Pat. No. US 5837493
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: INCYTE GENOMICS, INC., PATENT DEPARTMENT, 3160 Porter Drive, Palo Alto, CA, 94304
NUMBER OF CLAIMS: 47
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 2390
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides two novel human **galectins** (designated individually as GAL-5HA and GAL-5HB, and collectively as GAL-5H) and polynucleotides which identify and encode GAL-5H. The invention also provides genetically engineered **expression** vectors and host cells comprising the nucleic acid sequences encoding GAL-5H and a method for producing GAL-5H. The invention also provides for use of GAL-5H and agonists, antibodies, or antagonists specifically binding GAL-5H, in the prevention and treatment of diseases associated with **expression** of GAL-5H. Additionally, the invention provides for the use of antisense molecules to polynucleotides encoding GAL-5H for the treatment of diseases associated with the **expression** of GAL-5H. The invention also provides diagnostic assays which utilize the polynucleotide, or fragments or the complement thereof, and antibodies specifically binding GAL-5H.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 10 OF 31 USPATFULL
ACCESSION NUMBER: 2002:148596 USPATFULL
TITLE: Method and kit for predicting cancer
INVENTOR(S): Woo, Hee Jong, Kyonggi-Do, KOREA, REPUBLIC OF
PATENT ASSIGNEE(S): ZARITA BIOTECH CO., LTD., Kyonggi-do, KOREA, REPUBLIC

OF (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002076738	A1	20020620
APPLICATION INFO.:	US 2001-972356	A1	20011009 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	KR 2000-63868	20001030

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: STAAS & HALSEY LLP, 700 11TH STREET, NW, SUITE 500, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 16
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 865
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to a method and a kit for diagnosing and/or predicting the occurrence of cancer or the risk of contracting a cancer by measuring the concentration of a cancer screening antigen(CSA) in blood, which changes before the occurrence of the cancer in a patient. The method of diagnosing or predicting the occurrence of cancer or the risk of contracting a cancer comprising the steps of: determining a concentration of **galectin-3** in a blood sample by reacting the blood sample with a monoclonal antibody of the **galectin-3**; comparing the determined concentration of the **galectin-3** with concentration of the **galectin-3** in a blood sample of a normal human; and predicting the risk of contracting a cancer if the determined concentration is greater than the concentration of the **galectin -3** in blood of the normal human.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 11 OF 31 USPATFULL
ACCESSION NUMBER: 2002:22131 USPATFULL
TITLE: 18 Human secreted proteins
INVENTOR(S): Shi, Yanggu, Gaithersburg, MD, UNITED STATES
Young, Paul E., Gaithersburg, MD, UNITED STATES
Ebner, Reinhard, Gaithersburg, MD, UNITED STATES
Soppet, Daniel R., Centreville, VA, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002012966	A1	20020131
APPLICATION INFO.:	US 2001-768826	A1	20010125 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 2000-US22350, filed on 15 Aug 2000, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-148759P	19990816 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
LINE COUNT: 18157
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes

encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 12 OF 31 USPATFULL

ACCESSION NUMBER:	2002:275918 USPATFULL
TITLE:	**Galectin** 9 and 10SV polynucleotides
INVENTOR(S):	Ni, Jian, Rockville, MD, United States Gentz, Reiner L., Silver Spring, MD, United States Ruben, Steven M., Olney, MD, United States
PATENT ASSIGNEE(S):	Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6468768	B1	20021022
APPLICATION INFO.:	US 2000-656450		20000906 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1999-263689, filed on 5 Mar 1999 Division of Ser. No. US 1997-946914, filed on 9 Oct 1997, now patented, Pat. No. US 6027916

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-28093P	19961009 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	GRANTED
PRIMARY EXAMINER:	Caputa, Anthony C.
ASSISTANT EXAMINER:	Davis, Natalie
LEGAL REPRESENTATIVE:	Sterne, Kessler, Goldstein & Fox P.L.L.C.
NUMBER OF CLAIMS:	12
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	18 Drawing Figure(s); 18 Drawing Page(s)
LINE COUNT:	2945

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel **galectin** 8, 9, 10 and 10SV proteins which are members of the **galectin** superfamily. In particular, isolated nucleic acid molecules are provided encoding the human **galectin** 8, 9, 10 and 10SV proteins. **Galectin** 8, 9, 10 and 10SV polypeptides are also provided as are vectors, host cells and recombinant methods for producing the same. The invention further relates to screening methods for identifying agonists and antagonists of **galectin** 8, 9, 10 or 10SV activity. Also provided are diagnostic and therapeutic methods.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 13 OF 31 USPATFULL

ACCESSION NUMBER:	2002:181517 USPATFULL
TITLE:	Method of diagnosing juvenile polyposis (JP)
INVENTOR(S):	Howe, James R., Iowa City, IA, United States Aaltonen, Lauri A., Espoo, FINLAND
PATENT ASSIGNEE(S):	University of Iowa Research Foundation, Iowa City, IA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6423491	B1	20020723
APPLICATION INFO.:	US 1999-312748		19990513 (9)

	NUMBER	DATE

PRIORITY INFORMATION: US 1998-85312P 19980513 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Caputa, Anthony C.
ASSISTANT EXAMINER: Harris, Alana M.
LEGAL REPRESENTATIVE: Fulbright & Jaworski, LLP
NUMBER OF CLAIMS: 30
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Figure(s); 5 Drawing Page(s)
LINE COUNT: 4588
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Familial juvenile polyposis is an autosomal dominant disease characterized by a predisposition to hamartomatous polyps and gastrointestinal cancer. The present invention shows that JP families carry germline mutations in SMAD4/DPC4, a gene on chromosome 18q21.1. The mutant SMAD4 proteins are truncated at the carboxyl-terminus and lack sequences required for normal function. Methods and compositions for the detection and amelioration of FJP and gastrointestinal tumors are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 14 OF 31 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:927718 CAPLUS
DOCUMENT NUMBER: 138:12503
TITLE: Mammalian **diabetes**-mediating proteins identification for diagnosis and therapy
INVENTOR(S): Larsen, Peter Mose; Fey, Stephen J.; Karlsen, Allan E.; Sparre, Thomas; Nerup, Jorn
PATENT ASSIGNEE(S): Syddansk Universitet, Den.
SOURCE: PCT Int. Appl., 128 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002097441	A2	20021205	WO 2002-DK368	20020529

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: DK 2001-852 A 20010529
DK 2002-446 A 20020322

AB Provided are mammalian secreted and non-secreted **diabetes** mediating proteins, including protective and deleterious **diabetes** -mediating proteins, as well as polynucleotides encoding same, drug screening methods for identifying a test compd. capable of altering the **expression** of a **diabetes**-mediating protein, and methods of preventing or ameliorating **diabetes** by administering a compd. capable of altering the **expression** of a **diabetes** -mediating protein. The proteins were identified by monitoring IL-1.beta. induced protein changes in **diabetes** prone mammalian islets of Langerhans using two-dimensional gel electrophoresis. Protein spots that significantly changed **expression** levels after exposure to IL-1.beta. were cut out of the gels and subjected to MALDI mass spectrometry. Eighty-two significantly changed protein spots were

detected. Pos. identification was obtained for a total of 45 different proteins from 51 of the 82 spots.

L44 ANSWER 15 OF 31 BIOTECHDS COPYRIGHT 2003 THOMSON DERWENT AND ISI
ACCESSION NUMBER: 2002-15178 BIOTECHDS
TITLE: Identifying anchor proteins that bind Ras protein, by producing complexes of Ras and cell membrane proteins in the presence and absence of a Ras antagonist and identifying a complex disrupted by the Ras antagonist;
antisense oligonucleotide transfer and **expression** in host cell for drug screening and gene therapy
AUTHOR: KLOOG Y; HAKLAI R; PAZ A; EL AD-SFADIA G; BALLAN E
PATENT ASSIGNEE: UNIV RAMOT APPLIED RES and IND DEV LTD
PATENT INFO: WO 2002029031 11 Apr 2002
APPLICATION INFO: WO 2000-IL918 4 Oct 2000
PRIORITY INFO: US 2000-237858 4 Oct 2000
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2002-435333 [46]
AN 2002-15178 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - Identifying (M1) cell membrane anchor proteins that bind a Ras protein (RP), involves preparing 2 reaction mixtures comprising RP, its cell membranes or fragments, where one mixture has a Ras antagonist, adding a cross linking agent, where complexes (C) between RP and other proteins are produced, separating (C), identifying (C), and separating RP from other proteins in (C), is new.

DETAILED DESCRIPTION - Identifying (M1) cell membrane anchor proteins that bind a Ras protein (RP), comprises: (a) preparing a first reaction mixture comprising RP, its cell membranes or fragments, and a second reaction mixture comprising RP and its cell membranes or fragments but not the Ras antagonist; (b) adding a cross-linking agent to the first and second reaction mixture, where cross linked (C) between RP and other proteins are produced; (c) separating each of the cross-linked (C) individually; (d) identifying (C) formed in the second reaction mixture that is disrupted by the Ras antagonist present in the first reaction mixture; and (e) separating the identified (C) from the other (C), and separating RP from the other protein in the separated (C) INDEPENDENT CLAIMS are also included for the following: (1) Identifying (M2) drug candidates that inhibit aberrant Ras activity, by preparing a reaction mixture containing RP, an anchor protein that binds RP and the drug candidate, and determining the effect of the drug candidate on interaction between RP and the anchor protein; (2) determining effective dosages of Ras antagonist that disrupt Ras-anchor protein binding, by contacting cells with the antagonist in-vivo or in-vitro, collecting the cells after contacting the cells, isolating cell membranes from the collected cells, measuring the decrease in anchor protein concentration per unit of cell membrane protein, and correlating the decrease with dosage of the Ras antagonist; (3) an antisense compound (AC) that specifically binds a nucleic acid encoding **galectin**-1, **galectin**-3, **galectin**-7 or **galectin**-8, and which causes degradation of the nucleic acid; (4) a composition comprising the above method.

BIOTECHNOLOGY - Preferred Method: In (M1), the Ras-antagonist is an inhibitor of prenylated or farnesylated RP, or is S-trans, trans-farnesylthiosalicylic acid (FTS) or its analog such as 5-fluoro-FTS, 5-chloro-FTS, 4-chloro-FTS, 2-chloro-5-farnesylaminobenzoic acid, farnesyl thionicotinic acid, S-farnesyl-methylthiosalicylic acid or 3-farnesylthio-cis-acrylic. The antagonist is an inhibitor of a non-prenylated RP. The cell membranes are obtained from NIH fibroblasts transformed with oncogenic K-Ras 4B (12V), H-Ras (12V) or N-Ras (13V), 518A2/N-Ras melanoma cells, 607B melanoma cells, Panc-1 cells containing oncogenic K-Ras, EJ cells containing H-Ras (12V) or MC-MA-11 cells. The cross linking agent is disuccininimidyl subarate (DSS), or dithiobis succinimdyl proprionate (DSP). In (M2), the effect of the drug candidate

is determined by measuring change in extent of dimerization of RP, or binding of Raf protein to RP, or binding between RP and anchor protein, or the change in activation of Raf protein. The reaction mixture further comprises a cross-linking agent. RP or the anchor protein is immobilized on a matrix. The anchor and RP are in solution and are detectably labeled with a fluorescent protein (FP) such as green or yellow FP. The anchor protein comprises **galectin**-1, **galectin-3**, **galectin**-7 or **galectin**-8. RP and the anchor protein are provided in the form of living cells. Determination comprises measuring loss of RP from the anchor protein, and observing intracellular movement of RP or the anchor protein.

ACTIVITY - Cytostatic; Immunosuppressive; Antidiabetic; Antiatherosclerotic; Neuroprotective; Vasotropic; Hepatotropic. No suitable data given.

MECHANISM OF ACTION - Antisense therapy.

USE - M1 is useful for identifying a cell membrane anchor protein that binds a Ras protein. M2 is useful for identifying drug candidates that inhibit aberrant Ras activity. AC comprising at least one phosphorathioate-modified nucleotide is useful for disrupting aberrant Ras activity in vivo, by infusing AC into a patient exhibiting this problem (claimed). M1 is also useful for identifying anchor proteins for the farnesylated isoforms of H-Ras, K-Ras 4A, K-Ras 4B and N-Ras, whose mutated forms are known to be oncogenic. Reducing or inhibiting aberrant Ras activity in vivo is useful for treating diseases characterized by uncontrolled mitosis, including cancers and various non-malignancies such as autoimmune disease (e.g. type 1 **diabetes**, lupus and multiple sclerosis), cirrhosis, graft rejection, atherosclerosis, polycystic kidneys and post-angioplasty restenosis.

ADMINISTRATION - AC is administered to the patient by a liposome, at a dose of 0.06-7 mg/kg/day. No administration routes given.

EXAMPLE - Chemical cross-linkers were used to identify the rapidly dissociating complexes of Ras and Tas-interacting proteins. The Ras inhibitor farnesylthiosalicylic acid (FTS) was used as an analytical tool to identify complexes sensitive to this inhibitor. The analytical steps were performed with controls and with FTS-treated EJ cells in combination with the cross-linkers disuccininimidyl subarate (DSS) and dithiobis succinimdyl proprionate (DSP). When membranes of control and FTS-treated cells were exposed to these cross-linkers solubilized and fractionated on sodium dodecyl sulfate-containing gels, Ras-immunoreactive bands were clearly detected at 34-43, 50 and 70 kDa. These complexes were not detected in the absence of the cross linkers. The broad band at 34-43 was not present in cells or cell membranes after treatment with FTS. Interaction of Ras with the IDRAs was not disrupted with analogs of FTS that had no anti-Ras activity on tumor cells. Triton X-100 extracts of the membranes containing Ras complexes formed by cross-linking with DSP were used for subsequent purification. FPLC MonoQ ion exchange chromatography yielded an enriched preparation of Ras-protein complexes. Ras and all species of the Ras-immunoreactive complexes detected in the pooled MonoQ fractions were specifically immunoprecipitated by biotin-pan Ras antibody. Assuming that the larger complexes may represent multiples of the 34-43 kDa complexes, the Ras-immunoreactive band with the lowest molecular weight was further purified. Two consecutive gel purification steps were used. Under non-reducing conditions only the 34-43 kDa Ras-immunoreactive band was detected by Western immunoblotting with Ras antibody and 21 kDa Ras was released from the complexes by reduction with dithiothreitol. In addition, two major proteins were released by DTT from the 34-43 kDa Ras-immunoreactive complexes. One was a 14-15 kDa and the other a 19-20 kDa protein. The amounts of both of these were significantly lower in Rat-1 cells compared to EJ cells. The 14-15 kDa protein was barely detected in the myr H-Ras (12V) cells. These results suggested that the 14-15 kDa protein interacted with the farnesylated H-Ras and was involved in cell transformation induced by this Ras isoform. The highly purified protein released by reduction was subjected to trypsin cleavage followed by microbore high performance liquid chromatography (HPLC) separation of the tryptic fragments and MS analysis

of the isolated peptides. Fragmentation patterns of two peptides corresponded precisely to the 14kDa rat **galectin**-1. The fact that **galectin** was a 14 kDa protein further confirmed that the FTS-sensitive Ras-interacting protein was **galectin**-1, a previously identified sugar binding protein. (62 pages)

L44 ANSWER 16 OF 31 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 2
ACCESSION NUMBER: 2002:877818 CAPLUS
DOCUMENT NUMBER: 138:151940
TITLE: IL-1.beta. induced protein changes in **diabetes** prone BB rat islets of Langerhans identified by proteome analysis
AUTHOR(S): Sparre, T.; Bjerre Christensen, U.; Mose Larsen, P.; Fey, S. J.; Wrzesinski, K.; Roepstorff, P.; Mandrup-Poulsen, T.; Pociot, F.; Karlsen, A. E.; Nerup, J.
CORPORATE SOURCE: Steno Diabetes Center, Gentofte, DK-2820, Den.
SOURCE: Diabetologia (2002), 45(11), 1550-1561
CODEN: DBTGAJ; ISSN: 0012-186X
PUBLISHER: Springer-Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Aims/hypothesis. Type I (insulin-dependent) **diabetes** mellitus is characterized by selective destruction of the insulin producing beta cells. Interleukin-1.beta. (IL-1.beta.) modulates the beta-cell function, protein synthesis, energy prodn. and causes apoptosis. We have previously shown changes in the **expression** of 82 out of 1 815 protein spots detected by two dimensional gel electrophoresis in IL-1.beta. exposed **diabetes** prone Bio Breeding (BB-DP) rat islets of Langerhans in vitro. The aim of this study was to identify the proteins in these 82 spots by mass spectrometry and compare these changes with those seen in IL-1.beta. exposed Wistar Furth (WF) rat islets. Methods. The 82 protein spots, that changed **expression** after IL-1.beta. exposure, were all re-identified on preparative gels of 200 000 neonatal WF rat islets, cut out and subjected to mass spectrometry for identification. Results. Forty-five different proteins were identified from 51 spots and grouped according to function: (i) energy transduction and redox potentials; (ii) glycolytic and Krebs cycle enzymes; (iii) protein, DNA and RNA synthesis, chaperoning and protein folding; (iv) signal transduction, regulation, differentiation and apoptosis; (v) cellular defense; and (vi) other functions. Comparison of IL-1.beta. exposed BB-DP and WF islets showed common changes in 14 proteins and several proteins influencing similar pathways, suggesting that similar routes in the two strains lead to beta-cell destruction. Conclusion/interpretation. We demonstrate that proteome anal. is a powerful tool to identify proteins and pathways in BB-DP rat islets exposed to IL-1.beta..
REFERENCE COUNT: 97 THERE ARE 97 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L44 ANSWER 17 OF 31 USPATFULL
ACCESSION NUMBER: 2001:185052 USPATFULL
TITLE: Modulators of leaderless protein export and methods for identifying and using the same
INVENTOR(S): Florkiewicz, Robert Z., Ramona, CA, United States
Baird, Andrew, San Diego, CA, United States
Warnock, Dale E., San Diego, CA, United States
PATENT ASSIGNEE(S): Ciblex Corporation, San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6306613	B1	20011023
APPLICATION INFO.:	US 1999-451905		19991201 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1998-30613, filed on 25 Feb 1998, now patented, Pat. No. US 6083706

Continuation-in-part of Ser. No. US 1997-807014, filed on 26 Feb 1997
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Smith, Lynette R. F.
ASSISTANT EXAMINER: Baskar, Padma
LEGAL REPRESENTATIVE: Seed Intellectual Property Law Group PLLC
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 47 Drawing Figure(s); 41 Drawing Page(s)
LINE COUNT: 2896
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods of modulating the export of a leaderless protein from a cell by contacting the cell with a compound that alters the binding of the leaderless protein and a transport molecule are provided. Transport molecules include gastrin binding protein/alpha subunit of mitochondrial fatty acid .beta.-oxidation multienzyme complex (p70, GenBank Accession Nos. U04627/D16480), phosphotyrosine-independent ligand of the SH2 domain of p56lck (p62, GenBank Accession No. U46751), mitochondrial fatty acid .beta.-oxidation trifunctional protein .beta. subunit (TP-.beta.) (p48, GenBank Accession No. D16481), actin related protein 3 (Arp3) (p48, GenBank Accession No. U29610), K-glypican (GenBank Accession No. X83577), tubulin (p50, GenBank Accession No. AF081484) and related polypeptides that are functionally equivalent in their role as leaderless protein trafficking components. Leaderless proteins include, for example, FGF-1, FGF-2, IL-1.alpha., IL-1.beta., CNTF, MIF, and HIV tat. These methods are useful in treatment of various conditions, including tumors and **diabetes** as well is in identifying small molecules for export modulation.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 18 OF 31 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 3
ACCESSION NUMBER: 2001:111081 CAPLUS
DOCUMENT NUMBER: 134:293886
TITLE: CD36, a member of the class B scavenger receptor family, as a receptor for advanced glycation end products
AUTHOR(S): Ohgami, Nobutaka; Nagai, Ryoji; Ikemoto, Mamoru; Arai, Hiroyuki; Kuniyasu, Akihiko; Horiuchi, Seikoh; Nakayama, Hitoshi
CORPORATE SOURCE: Department of Biofunctional Chemistry, Faculty of Pharmaceutical Sciences, Kumamoto University, Kumamoto, 862-0973, Japan
SOURCE: Journal of Biological Chemistry (2001), 276(5), 3195-3202
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Interaction of advanced glycation end products (AGE) with AGE receptors induces several cellular phenomena potentially relating to diabetic complications. Five AGE receptors identified so far are RAGE (receptor for AGE), **galectin-3**, 80K-H, OST-48, and SRA (macrophage scavenger receptor class A types I and II). Since SRA is known to belong to the class A scavenger receptor family, and the scavenger receptor collectively represents a family of multiligand lipoprotein receptors, it is possible that CD36, although belonging to the class B scavenger receptor family, can recognize AGE proteins as ligands. This was tested at the cellular level in this study using Chinese hamster ovary (CHO) cells overexpressing human CD36 (CD36-CHO cells). Cellular **expression** of CD36 was confirmed by immunoblotting and immunofluorescent microscopy using anti-CD36 antibody. Upon incubation at 37.degree., 125I-AGE-bovine serum albumin (AGE-BSA) and 125I-oxidized low

d. lipoprotein (LDL), an authentic ligand for CD36, were endocytosed in a dose-dependent fashion and underwent lysosomal degrdn. by CD36-CHO cells, but not wild-type CHO cells. In binding expts. at 4.degree., 125I-AGE-BSA exhibited specific and saturable binding to CD36-CHO cells (Kd = 5.6 .mu.g/mL). The endocytic uptake of 125I-AGE-BSA by these cells was inhibited by 50% by oxidized LDL and by 60% by FA6-152, an anti-CD36 antibody inhibiting cellular binding of oxidized LDL. The authors' results indicate that CD36 expressed by these cells mediates the endocytic uptake and subsequent intracellular degrdn. of AGE proteins. Since CD36 is one of the major oxidized LDL receptors and is up-regulated in macrophage- and smooth muscle cell-derived foam cells in human atherosclerotic lesions, these results suggest that, like oxidized LDL, AGE proteins generated in situ are recognized by CD36, which might contribute to the pathogenesis of diabetic macrovascular complications.
REFERENCE COUNT: 73 THERE ARE 73 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L44 ANSWER 19 OF 31 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 2001643880 MEDLINE
DOCUMENT NUMBER: 21548007 PubMed ID: 11689472
TITLE: Accelerated diabetic glomerulopathy in **galectin-3**/AGE receptor 3 knockout mice.
AUTHOR: Pugliese G; Pricci F; Iacobini C; Leto G; Amadio L; Barsotti P; Frigeri L; Hsu D K; Vlassara H; Liu F T; Di Mario U
CORPORATE SOURCE: Department of Clinical Sciences, 'La Sapienza' University, 00161 Rome, Italy.. giuseppe.pugliese@uniroma1.it
SOURCE: FASEB JOURNAL, (2001 Nov) 15 (13) 2471-9.
Journal code: 8804484. ISSN: 1530-6860.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011107
Last Updated on STN: 20020123
Entered Medline: 20011205
AB Several molecules were shown to bind advanced glycation end products (AGEs) in vitro, but it is not known whether they all serve as AGE receptors and which functional role they play in vivo. We investigated the role of **galectin-3**, a multifunctional lectin with (anti)adhesive and growth-regulating properties, as an AGE receptor and its contribution to the development of diabetic glomerular disease, using a knockout mouse model. **Galectin-3** knockout mice obtained by gene ablation and the corresponding wild-type mice were rendered diabetic with streptozotocin and killed 4 months later, together with age-matched nondiabetic controls. Despite a comparable degree of metabolic derangement, **galectin-3**-deficient mice developed accelerated glomerulopathy vs. the wild-type animals, as evidenced by the more pronounced increase in proteinuria, extracellular matrix gene **expression**, and mesangial expansion. This was associated with a more marked renal/glomerular AGE accumulation, indicating it was attributable to the lack of **galectin-3** AGE receptor function. The **galectin-3**-deficient genotype was associated with reduced **expression** of receptors implicated in AGE removal (macrophage scavenger receptor A and AGE-R1) and increased **expression** of those mediating cell activation (RAGE and AGE-R2). These results show that the **galectin-3** -regulated AGE receptor pathway is operating in vivo and protects toward AGE-induced tissue injury in contrast to that through RAGE.

L44 ANSWER 20 OF 31 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 5
ACCESSION NUMBER: 2001:306914 CAPLUS
DOCUMENT NUMBER: 134:293560
TITLE: AGE and AGE-receptors

AUTHOR(S): Ohgami, Nobutaka; Nagai, Ryoji; Nakayama, Hitoshi; Horiuchi, Seikoh
CORPORATE SOURCE: Dep. Siofunctional Chem., Fac. Pharm. Sci., Kumamoto Univ., 5-1, Oe-honmachi, Kumamoto, 862-0973, Japan
SOURCE: Seikagaku (2001), 73(3), 200-204
CODEN: SEIKAQ; ISSN: 0037-1017
PUBLISHER: Nippon Seikagakkai
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review with 15 refs., on advanced glycation end products (AGE) and its receptors involved in aging-related diabetic complications and atherosclerosis, discussing AGE formation and its relevance to aging, class A scavenger receptors involved in AGE clearance, **expression** and functions of AGE-binding proteins (OST-48, 80K-H, and **galectin -3**), function of RAGE (receptor for AGE), and physiol. significance of a novel AGE receptor, CD36, belonging to the scavenger receptor family.

L44 ANSWER 21 OF 31 USPATFULL
ACCESSION NUMBER: 2000:21410 USPATFULL
TITLE: **Galectin** 9 and 10SV Polynucleotides
INVENTOR(S): Ni, Jian, Rockville, MD, United States
Gentz, Reiner L., Silver Spring, MD, United States
Ruben, Steven M., Olney, MD, United States
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6027916		20000222	
APPLICATION INFO.:	US 1997-946914		19971009	(8)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-28093P	19961009 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Hutzell, Paula K.
ASSISTANT EXAMINER: Sun-Hoffman, Lin
LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox, P.L.L.C.
NUMBER OF CLAIMS: 34
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Figure(s); 18 Drawing Page(s)
LINE COUNT: 3299
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to novel **galectin** 8, 9, 10 and 10SV proteins which are members of the **galectin** superfamily. In particular, isolated nucleic acid molecules are provided encoding the human **galectin** 8, 9, 10 and 10SV proteins. **Galectin** 8, 9, 10 and 10SV polypeptides are also provided as are vectors, host cells and recombinant methods for producing the same. The invention further relates to screening methods for identifying agonists and antagonists of **galectin** 8, 9, 10 or 10SV activity. Also provided are diagnostic and therapeutic methods.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 22 OF 31 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 6
ACCESSION NUMBER: 2000:477959 CAPLUS
DOCUMENT NUMBER: 133:191437
TITLE: The diabetic milieu modulates the advanced glycation end product-receptor complex in the mesangium by inducing or upregulating **galectin-3 expression**
AUTHOR(S): Pugliese, Giuseppe; Pricci, Flavia; Leto, Gaetano;

Amadio, Lorena; Iacobini, Carla; Romeo, Giulio; Lenti, Luisa; Sale, Patrizio; Gradini, Roberto; Liu, Fu-Tong; Di Mario, Umberto

CORPORATE SOURCE: Department of Clinical Sciences, La Sapienza University, Rome, Italy

SOURCE: Diabetes (2000), 49(7), 1249-1257
CODEN: DIAEAZ; ISSN: 0012-1797
PUBLISHER: American Diabetes Association
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Nonenzymic glycation has been implicated in the pathogenesis of the dysregulated tissue remodeling that characterizes diabetic glomerulopathy, via the formation of advanced glycation end products (AGEs) and their binding to cell surface receptors. Several AGE-binding proteins have been identified so far, including p60, p90, and the adhesive and growth-regulating lectin **galectin-3** (Gal-3), the components of the so-called AGE-receptor complex. This study aimed to evaluate the mesangial **expression** of the AGE-receptor complex and its modulation by the diabetic milieu, both in vivo, in non-diabetic vs. streptozotocin-induced diabetic rats, and in vitro, in mesangial cells exposed to either normal glucose (NG) levels (5.5 mmol/l), as compared with high glucose (HG) levels (30 mmol/l) and iso-osmolar mannitol (M), or to native bovine serum albumin (BSA), as compared with glycated BSA with AGE formation (BSA-AGE) and glycated BSA in which AGE formation was prevented by aminoguanidine (BSA-AM). In vivo, Gal-3 protein and mRNA were not detectable in glomeruli from nondiabetic rats until 12 mo after initiating the study. On the contrary, in diabetic rats, Gal-3 **expression** was obsd. at 2 mo of disease duration, and it increased thereafter. Both p60 and p90 immunoreactivities were obsd. at the glomerular level with slightly increased **expression** of p90, but not p60, in diabetic vs. nondiabetic animals. In vitro, Gal-3 was not detectable in mesangial cells cultured in NG (although it became evident after a certain no. of passages in culture), whereas Gal-3 was detectable in cells grown on BSA. Prolonged exposure (2-4 wk) of mesangial cells to HG but not to M, as well as growing cells on BSA-AGE and, to a lesser extent, BSA-AM, induced or significantly increased the **expression** of Gal-3, both protein (up to 2.65-fold) and mRNA (up to 3.10-fold) and its secretion in the medium (by .apprx.50%). Both p60 and p90 were demonstrated in mesangial cells under NG conditions, and the **expression** of p90, but not p60, was upregulated by .apprx.20% by HG or BSA-AGE. These results indicate that (1) under basal conditions, Gal-3, unlike p90 and p60, is not detectable in the mesangium but becomes expressed with aging and (2) the diabetic milieu induces or upregulates Gal-3 prodn., whereas it increases only slightly the **expression** of p90, but not p60. Gal-3 **expression** or overexpression may modulate the AGE-receptor-mediated events by modifying the function of the AGE-receptor complex. Addnl., it may exert direct effects on tissue remodeling by virtue of its adhesive and growth-regulating properties.

REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L44 ANSWER 23 OF 31 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 7

ACCESSION NUMBER: 2000:667186 CAPLUS
DOCUMENT NUMBER: 133:347635

TITLE: Role of **galectin-3** as a receptor for advanced glycosylation end products

AUTHOR(S): Pricci, Flavia; Leto, Gaetano; Amadio, Lorena; Iacobini, Carla; Romeo, Giulio; Cordone, Samantha; Gradini, Roberto; Barsotti, Paola; Liu, Fu-Tong; Di Mario, Umberto; Pugliese, Giuseppe

CORPORATE SOURCE: Department of Clinical Sciences, Division of Endocrinology, "La Sapienza" University, Rome, Italy

SOURCE: Kidney International, Supplement (2000), 77, S31-S39
CODEN: KISUDF; ISSN: 0098-6577
PUBLISHER: Blackwell Science, Inc.

DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 90 refs. The advanced glycosylation end product (AGE)-binding proteins identified so far include the components of the AGE-receptor complex p60, p90 and **galectin-3**, receptor for advanced glycosylation end products (RAGE), and the macrophage scavenger receptor types I and II. **Galectin-3** interacts with .beta.-galactoside residues of several cell surface and matrix glycoproteins through the carbohydrate recognition domain and is also capable of peptide-peptide assocns. mediated by its N-terminus domain. These structural properties enable **galectin-3** to exert multiple functions, including the modulation of cell adhesion, the control of cell cycle, and the mRNA splicing activity. Moreover, in macrophages, astrocytes, and endothelial cells, **galectin-3** has been shown to exhibit a high-affinity binding for AGEs; the lack of a transmembrane anchor sequence or signal peptide suggests that it assocs. with other AGE-receptor components rather than playing an independent role as AGE-receptor. In tissues that are targets of diabetic vascular complications, such as the mesangium and the endothelium, **galectin-3** is not expressed or only weakly expressed under basal conditions, at variance with p90 and p60 but becomes detectable with aging and is induced or up-regulated by the diabetic milieu, which only slightly affects the **expression** of p90 or p60. This (over)**expression** of **galectin-3** may in turn modulate AGE-receptor-mediated events by modifying the function of the AGE-receptor complex, which could play a role in the pathogenesis of target tissue injury. Up-regulated **galectin-3 expression** may also exert direct effects on tissue remodeling, independently of AGE ligands, by virtue of its adhesive and growth regulating properties.
REFERENCE COUNT: 90 THERE ARE 90 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L44 ANSWER 24 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:511195 BIOSIS
DOCUMENT NUMBER: PREV199900511195
TITLE: Renal tubular **expression** of endogenous 31kD **galectin** is increased in diabetic kidney.
AUTHOR(S): Patel, Sharmila (1); Zhang, Zhi (1); Yamaoka, Kazuyoshi (1); Hughes, R. Colin; Hartley, Barrie; Showaya, Suhier E. I.; Porter, Christine J.; Cassidy, M. J. D.; Williams, David G. (1)
CORPORATE SOURCE: (1) MRC Collaborative Centre, London UK
SOURCE: Journal of the American Society of Nephrology, (Sept., 1999) Vol. 10, No. PROGRAM AND ABSTR. ISSUE, pp. 578A. Meeting Info.: 32nd Annual Meeting of the American Society of Nephrology Miami Beach, Florida, USA November 1-8, 1999 American Society of Nephrology . ISSN: 1046-6673.
DOCUMENT TYPE: Conference
LANGUAGE: English

L44 ANSWER 25 OF 31 USPATFULL
ACCESSION NUMBER: 1998:143897 USPATFULL
TITLE: Human **galectins**
INVENTOR(S): Hillman, Jennifer L., San Jose, CA, United States
Goli, Surya K., Sunnyvale, CA, United States
Bandman, Olga, Mountain View, CA, United States
Hawkins, Phillip R., Mountain View, CA, United States
Petithory, Joanne R., Fremont, CA, United States
PATENT ASSIGNEE(S): Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)

NUMBER	KIND	DATE

PATENT INFORMATION: US 5837493 19981117
APPLICATION INFO.: US 1997-788584 19970123 (8)
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Feisee, Lila
ASSISTANT EXAMINER: Sun-Hoffman, Lin
LEGAL REPRESENTATIVE: Billings, Lucy J.Incyte Pharmaceuticals, Inc.
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Figure(s); 7 Drawing Page(s)
LINE COUNT: 2242
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides two novel human **galectins** (designated individually as GAL-5HA and GAL-5HB, and collectively as GAL-5H) and polynucleotides which identify and encode GAL-5H. The invention also provides genetically engineered **expression** vectors and host cells comprising the nucleic acid sequences encoding GAL-5H and a method for producing GAL-5H. The invention also provides for use of GAL-5H and agonists, antibodies, or antagonists specifically binding GAL-5H, in the prevention and treatment of diseases associated with **expression** of GAL-5H. Additionally, the invention provides for the use of antisense molecules to polynucleotides encoding GAL-5H for the treatment of diseases associated with the **expression** of GAL-5H. The invention also provides diagnostic assays which utilize the polynucleotide, or fragments or the complement thereof, and antibodies specifically binding GAL-5H.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L44 ANSWER 26 OF 31 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:324881 CAPLUS
DOCUMENT NUMBER: 129:39786
TITLE: **Diabetes**-mediating proteins and their therapeutic uses
INVENTOR(S): Mose, Larsen Peter; Fey, Stephen J.; Nerup, Jorn; Karlsen, Allan E.; Bjerre, Christensen Ulla; Pociot, Flemming; Andersen, Henrik U.
PATENT ASSIGNEE(S): Mose Larsen, Peter, Den.; Fey, Stephen J.; Nerup, Jorn; Karlsen, Allan E.; Bjerre Christensen, Ulla; Pociot, Flemming; Andersen, Henrik U.
SOURCE: PCT Int. Appl., 145 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9820124	A2	19980514	WO 1997-IB1627	19971024
WO 9820124	A3	19981008		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
JP 2001500614	T2	20010116	JP 1998-513441	19970916
AU 9854070	A1	19980529	AU 1998-54070	19971024
EP 934409	A2	19990811	EP 1997-947839	19971024
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001503860	T2	20010321	JP 1998-520234	19971024

JP 2002504806	T2	20020212	JP 1998-521182		19971024
KR 2000052802	A	20000825	KR 1999-703621		19990424
PRIORITY APPLN. INFO.:			US 1996-29324P	P	19961025
			US 1996-30088P	P	19961105
			US 1996-30186P	P	19961105
			US 1997-897098	A2	19970718
			US 1996-31291P	P	19960916
			US 1996-29325P	P	19961025
			WO 1997-IB1114	W	19970916
			WO 1997-IB1337	W	19971024
			WO 1997-IB1627	W	19971024

AB Protective and deleterious **diabetes**-mediating proteins involved in the development of **diabetes** or in the prevention of **diabetes** development are identified by differential **expression** during during development of **diabetes** relative to **expression** in the absence of **diabetes** development. These proteins are referred to by their position on 10% IEF or NEPHGE 2-dimensional gels. The purified **diabetes**-mediating proteins are characterized by mol. wt., isoelec. point, and mass spectroscopic characteristics. **Galectin**-3 (rat and human) and mortalin (mouse and human), two of the identified proteins from pancreatic islets, were also sequenced. Transgenic animals expressing a **diabetes**-mediating protein, drug screening methods for identifying a test compd. capable of altering the **expression** of a **diabetes**-mediating protein, and methods of preventing or ameliorating **diabetes** by administering a compd. capable of altering the **expression** of a **diabetes**-mediating protein are also provided..

L44 ANSWER 27 OF 31 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 8

ACCESSION NUMBER:	1998:760707 CAPLUS
DOCUMENT NUMBER:	130:107885
TITLE:	Cell activation by glycated proteins. AGE receptors, receptor recognition factors and functional classification of AGEs
AUTHOR(S):	Thornalley, Paul J.
CORPORATE SOURCE:	Department of Biological Sciences, University of Essex, Essex, CO4 3SQ, UK
SOURCE:	Cellular and Molecular Biology (Paris) (1998), 44(7), 1013-1023 CODEN: CMOBEF; ISSN: 0145-5680
PUBLISHER:	C.M.B. Association
DOCUMENT TYPE:	Journal; General Review
LANGUAGE:	English

AB A review, with .apprx.72 refs. Proteins modified by advanced glycation end products (AGE) bind to cell surface receptors and other AGE binding proteins. AGE-binding receptors are: scavenger receptors types I and II, the receptor for advanced glycation end products (RAGE), oligosaccharyl transferase-48 (OST-48, AGE-R1), 80K-H phosphoprotein (AGE-R2) and **galectin-3** (AGE-R3). AGE receptors are found in monocytes, macrophages, endothelial cells, pericytes, podocytes, astrocytes and microglia. AGE-modified proteins also bind to lysozyme and lactoferrin. A crit. review of the evidence for receptors binding AGE-modified protein binding in vivo is presented. Scavenger receptors have only been shown to bind proteins modified by AGE to a much higher extent than found in vivo. 80K-H phosphoprotein is involved in FGFR3 signal transduction to MAP kinase, and may be involved in AGE-receptor signal transduction. Whether all of these proteins bind AGE-modified proteins in vivo is not yet clear. Cell activation in response to AGE-modified proteins is assocd. with increased **expression** of extracellular matrix proteins, vascular adhesion mols., cytokines and growth factors. Depending on the cell type and concurrent signaling, this is assocd. with chemotaxis, angiogenesis, oxidative stress, cell proliferation or programmed cell death (PCD). Receptor recognition factors for agonism at the AGE receptor have been little studied but to

date hydroimidazolones appear to be the most likely candidates. Pharmacol. inhibition of AGE receptor-mediated cell activation with specific antagonists may provide the basis for therapeutic intervention in diseases where AGE accumulation is a suspected etiol. factor vascular complications of **diabetes**, macrovascular disease, renal insufficiency and Alzheimer's disease.

REFERENCE COUNT: 72 THERE ARE 72 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L44 ANSWER 28 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:424372 BIOSIS
DOCUMENT NUMBER: PREV199800424372
TITLE: Induction of glomerular/mesangial **galectin-3**/age-receptor-3 **expression** by the diabetic milieu.
AUTHOR(S): Leto, G. (1); Pricci, F. (1); Romeo, G. (1); Catalano, S. (1); Amadio, L. (1); Diaz-Horta, O. (1); Sale, P. (1); Gradini, R. (1); Lenti, L. (1); Barsotti, P. (1); Frigeri, L.; Dimario, U. (1); Pugliese, G. (1)
CORPORATE SOURCE: (1) "La Sapienza" Univ., Rome Italy
SOURCE: Diabetologia, (Aug., 1998) Vol. 41, No. SUPPL. 1, pp. A27. Meeting Info.: 34th Annual Meeting of the European Association for the Study of Diabetes Barcelona, Spain September 11, 1998 European Association for the Study of Diabetes
. ISSN: 0012-186X.
DOCUMENT TYPE: Conference
LANGUAGE: English

L44 ANSWER 29 OF 31 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1997:372276 BIOSIS
DOCUMENT NUMBER: PREV199799671479
TITLE: Modulation of **Galectin-3**/age-receptor-3 **expression** by the diabetic milieu in cultured rat mesangial cells.
AUTHOR(S): Pugliese, G. (1); Pricci, F.; Romeo, G.; Leto, G.; Gradini, R.; Santangelo, C.; Lenti, L.; Cirulli, V.; Hayek, A.; Liu, F. T.; Frigeri, L.; Di Mario, U.
CORPORATE SOURCE: (1) Univ. Rome La Sapienza Italy
SOURCE: Diabetologia, (1997) Vol. 40, No. SUPPL. 1, pp. A508. Meeting Info.: 16th International Diabetes Federation Congress Helsinki, Finland July 20-25, 1997
ISSN: 0012-186X.
DOCUMENT TYPE: Conference; Abstract; Conference
LANGUAGE: English

L44 ANSWER 30 OF 31 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 9
ACCESSION NUMBER: 1995:944605 CAPLUS
DOCUMENT NUMBER: 124:83769
TITLE: Identification of **galectin-3** as a high-affinity binding protein for advanced glycation end products (AGE): a new member of the AGE-receptor complex
AUTHOR(S): Vlassara, Helen; Li, Yong Ming; Imani, Farhad; Wojciechowicz, Donald; Yang, Zhi; Liu, Fu-Tong; Cerami, Anthony
CORPORATE SOURCE: Picower Institute for Medical Research, Manhasset, NY, USA
SOURCE: Molecular Medicine (Cambridge, Massachusetts) (1995), 1(6), 634-46
CODEN: MOMEF3; ISSN: 1076-1551
PUBLISHER: Blackwell
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Advanced glycation end products (AGE), the reactive derivs. of nonenzymic

glucose-protein condensation reactions, are implicated in the multiorgan complications of **diabetes** and aging. An AGE-specific cellular receptor complex (AGE-R) mediating AGE removal as well as multiple biol. responses has been identified. By screening an **expression** library using antibody against a previously identified component of the AGE-R complex p90, a known partial cDNA clone was isolated with homol. to **galectin-3**, a protein of diverse identity, and member of the **galectin** family. To explore this finding, the nature of the interactions between **galectin-3** and AGE was studied using intact macrophage-like RAW 264.7 cells, membrane-assocd. and recombinant **galectin**-1 through -4, and model AGE-ligands (AGE-BSA, FFI-BSA). Among the members of this family (**galectin** -1 through 4), recombinant rat **galectin-3** was found to exhibit high-affinity 125I-AGE-BSA binding with saturable kinetics (kD 3.5.times.107 M-1) that was fully blocked by excess unlabeled naturally formed AGE-BSA or synthetic FFI-BSA, but only weakly inhibited by several known **galectin-3** ligands, such as lactose. In addn. to the p90, immunopptn. with anti-**galectin-3**, followed by 125I-AGE-BSA ligand blot anal. of RAW 264.7 cell exts., revealed **galectin-3** (28 and 32 kDa), as well as **galectin** -**3**-assocd. proteins (40 and 50 kDa) with AGE-binding activity. Interaction of **galectin-3** with AGE-BSA or FFI-BSA resulted in formation of SDS-, and .beta.-mercaptoethanol-insol., but hydroxylamine-sensitive high-mol. wt. complexes between AGE-ligand, **galectin-3**, and other membrane components. The findings point toward a mechanism by which **galectin-3** may serve in the assembly of AGE-R components and in the efficient cell surface attachment and endocytosis by macrophages of a heterogeneous pool of AGE moieties with diverse affinities, thus contributing to the elimination of these pathogenic substances.

L44 ANSWER 31 OF 31 DGENE (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: AAW61960 protein DGENE
TITLE: Identification of **diabetes**-mediating protein(s) - by transplanting insulin-secreting cells into host at risk of developing **diabetes** and analysing protein **expression** in transplanted cells
INVENTOR: Andersen H U; Bjerre C H R I S T E N S E N U; Fey S J; Karlsen A E; Mose L A R S E N P; Nerup J; Pociot F
PATENT ASSIGNEE: (ANDE-I)ANDERSEN H U.
(CHRI-I) BJERRE CHRISTENSEN U.
(FEYS-I) FEY S J.
(KARL-I) KARLSEN A E.
(LARS-I) MOSE LARSEN P.
(NERU-I) NERUP J.
(POCI-I) POCIOT F.
PATENT INFO: WO 9820124 A2 19980514 154p
APPLICATION INFO: WO 1997-IB1627 19971024
PRIORITY INFO: US 1997-897098 19970718
US 1996-29324 19961025
US 1996-30088 19961105
US 1996-30186 19961105
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 1998-286940 [25]
DESCRIPTION: DMP microsequence of **galectin-3** (peak 22).
AN AAW61960 protein DGENE
AB Sequences shown in AAW61956 to AAW61961 repersent microsequences of **diabetes**-mediating proteins (DMP) identified by the method of invention. The invention provides methods for in vivo identification of a **diabetes**- mediating protein by transplanting insulin-secreting cells into host at risk of developing **diabetes** and analysing protein **expression** in transplanted cells. The DMPs are useful in drug screening assays for identifying compounds capable of modulating

the development of **diabetes**, useful as therapeutic agents for the treatment or prevention of **diabetes**, and useful as targets of therapeutic agents capable of preventing or ameliorating **diabetes** by modulating the **expression** of the DMP. Changes in the **expression** of specific DMPs is diagnostically useful as indicator of the development of **diabetes**.

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=> index bioscience medicine meetings
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       CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE,
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L16 1 FILE CANCERLIT

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L20           1 FILE TOXCENTER
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L22           1 FILE WPIDS
L23           1 FILE NLDB

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L25           81 DUP REM L24 (36 DUPLICATES REMOVED)

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L25  ANSWER 1 OF 81  USPATFULL                          DUPLICATE 1
ACCESSION NUMBER:       2003:133463  USPATFULL
TITLE:                  Gene therapy for the prevention of autoimmune disease
INVENTOR(S):            Fathman, C. Garrison, Stanford, CA, UNITED STATES

                             NUMBER        KIND    DATE
                        ---------------- ------ --------
PATENT INFORMATION:     US 2003091548      A1   20030515
APPLICATION INFO.:      US 2002-263937     A1   20021002  (10)

                               NUMBER         DATE
                        ------------------ --------
PRIORITY INFORMATION:   US 2001-326668P    20011002 (60)
DOCUMENT TYPE:          Utility
FILE SEGMENT:           APPLICATION
LEGAL REPRESENTATIVE:   BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD,
                        SUITE 200, MENLO PARK, CA, 94025
NUMBER OF CLAIMS:       10
EXEMPLARY CLAIM:        1
NUMBER OF DRAWINGS:     4 Drawing Page(s)
LINE COUNT:             635
AB      Autoimmune disease is treated by the delivery of a suppressive agent to
        the site of disease. Delivery is accomplished by introducing an
        expression vector encoding the suppressive agent into cells
        targeted for such sites, and administering the genetically modified
        cells to the patient. Suppressive agents of particular interest include
        IL-4; and anti-CD3 antibodies, particularly single chain anti-CD3
        antibodies. Cells of interest for delivery include T cells and T cell
        hybridomas, where the T cell antigen receptor recognizes epitopes
        associated with the autoimmune disease,. Alternatively, dendritic cells
        are used as delivery vectors.


L25  ANSWER 2 OF 81  USPATFULL
ACCESSION NUMBER:       2003:127198  USPATFULL
TITLE:                  Death associated kinase containing ankyr in repeats
                        (DAKAR) and methods of use
INVENTOR(S):            Bird, Timothy A., Bainbridge Island, WA, UNITED STATES
                        Holland, Pamela M., Seattle, WA, UNITED STATES
                        Peschon, Jacques J., Seattle, WA, UNITED STATES
                        Virca, George D., Bellevue, WA, UNITED STATES

                             NUMBER        KIND    DATE
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PATENT INFORMATION:     US 2003087411      A1   20030508
APPLICATION INFO.:      US 2002-164080     A1   20020604  (10)
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	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-295959P	20010604 (60)
	US 2001-334362P	20011129 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: IMMUNEX CORPORATION, LAW DEPARTMENT, 51 UNIVERSITY STREET, SEATTLE, WA, 98101
NUMBER OF CLAIMS: 16
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 5574
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention relates to DAKAR, a new member of the serine/threonine kinase family, methods of making such polypeptides, and to methods of using them to treat conditions associated with apoptosis and epithelial proliferation and differentiation, as well as methods to identify compounds that alter DAKAR-associated cellular activities.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 3 OF 81 USPATFULL
ACCESSION NUMBER: 2003:120747 USPATFULL
TITLE: Blood cell deficiency treatment method
INVENTOR(S): Ahlem, Clarence N., San Diego, CA, UNITED STATES
Reading, Christopher, San Diego, CA, UNITED STATES
Frincke, James, San Diego, CA, UNITED STATES
Stickney, Dwight, Granite Bay, CA, UNITED STATES
Lardy, Henry A., Madison, WI, UNITED STATES
Marwah, Padma, Middleton, WI, UNITED STATES
Marwah, Ashok, Middleton, WI, UNITED STATES
Prendergast, Patrick T., Straffan, IRELAND

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003083231	A1	20030501
APPLICATION INFO.:	US 2002-87929	A1	20020301 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-675470, filed on 28 Sep 2000, PENDING Continuation-in-part of Ser. No. US 2001-820483, filed on 29 Mar 2001, PENDING Continuation-in-part of Ser. No. US 2000-535675, filed on 23 Mar 2000, PENDING Continuation-in-part of Ser. No. US 1999-449004, filed on 24 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-449184, filed on 24 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-449042, filed on 24 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-461026, filed on 15 Dec 1999, ABANDONED Continuation-in-part of Ser. No. US 2000-586673, filed on 1 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-586672, filed on 1 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 1999-414905, filed on 8 Oct 1999, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-161453P	19991025 (60)
	US 2001-272624P	20010301 (60)
	US 2001-323016P	20010911 (60)
	US 2001-340045P	20011130 (60)
	US 2001-328738P	20011011 (60)
	US 2001-338015P	20011108 (60)
	US 2001-343523P	20011220 (60)
	US 1999-126056P	19991019 (60)
	US 1999-124087P	19990311 (60)

	US 1998-109923P	19981124 (60)
	US 1998-109924P	19981124 (60)
	US 1998-110127P	19981127 (60)
	US 1998-112206P	19981215 (60)
	US 1999-145823P	19990727 (60)
	US 1999-137745P	19990603 (60)
	US 1999-140028P	19990616 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOLLIS-EDEN PHARMACEUTICALS, INC., 4435 EASTGATE MALL, SUITE 400, SAN DIEGO, CA, 92121
NUMBER OF CLAIMS: 45
EXEMPLARY CLAIM: 1
LINE COUNT: 19428
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to the use of compounds to treat a number of conditions, such as thrombocytopenia, neutropenia or the delayed effects of radiation therapy. Compounds that can be used in the invention include methyl-2,3,4-trihydroxy-1-O-(7,17-dioxoandrost-5-ene-3.beta.-yl)-.beta.-D-glucopyranosiduronate, 16.alpha.,3.alpha.-dihydroxy-5.alpha.-androstan-17-one or 3,7,16,17-tetrahydroxyandrost-5-ene, 3,7,16,17-tetrahydroxyandrost-4-ene,3,7,16,17-tetrahydroxyandrost-1-ene or 3,7,16,17-tetrahydroxyandrostane that can be used in the treatment method.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 4 OF 81 USPATFULL
ACCESSION NUMBER: 2003:113528 USPATFULL
TITLE: Biguanide and sulfonylurea formulations for the prevention and treatment of insulin resistance and type 2 diabetes mellitus
INVENTOR(S): Pearson, Don C., Lakewood, WA, UNITED STATES
Richardson, Kenneth T., Anchorage, AK, UNITED STATES
PATENT ASSIGNEE(S): ChronoRX, LLC, Anchorage, AK, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003078269	A1	20030424	
APPLICATION INFO.:	US 2002-93476	A1	20020307	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-278270P	20010322	(60)
	US 2001-278271P	20010322	(60)
	US 2001-278296P	20010322	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
NUMBER OF CLAIMS: 130
EXEMPLARY CLAIM: 1
LINE COUNT: 4927
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention describes formulations that include either metformin, sulfonylurea or a biguanide-sulfonylurea combination as one active ingredient in addition to specific, other active ingredients. The compositions and dosage forms of the invention are clinically useful as methods for increasing the effectiveness, efficiency and safety of the included biguanide (metformin) and/or sulfonylurea in the prevention and treatment of insulin resistance and diabetes mellitus. The carefully chosen additional active ingredients of the invention are designed in a modular fashion to prevent and rectify adverse events associated with insulin resistance syndrome and diabetes mellitus, and those adverse

incidences associated with the concurrent use of metformin and/or the sulfonylureas. When clinically administered, the invention will provide therapeutic levels of metformin and of a sulfonylurea, alone or in combination, and broaden their usefulness. The invention will retard the progression of insulin resistance to type 2 diabetes, and reduce the serious microvascular and macrovascular complications commonly associated with insulin resistance syndrome and diabetes mellitus.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 5 OF 81 USPATFULL
ACCESSION NUMBER: 2003:112605 USPATFULL
TITLE: Formulations for the prevention and treatment of insulin resistance and type 2 diabetes mellitus
INVENTOR(S): Richardson, Kenneth T., Anchorage, AK, UNITED STATES
Pearson, Don C., Lakewood, WA, UNITED STATES
PATENT ASSIGNEE(S): ChronoRX LLC, Anchorage, AK (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003077335	A1	20030424	
APPLICATION INFO.:	US 2001-33730	A1	20011102	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2000-245471P	20001103	(60)
	US 2000-245950P	20001103	(60)
	US 2000-256033P	20001213	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
NUMBER OF CLAIMS: 104
EXEMPLARY CLAIM: 1
LINE COUNT: 4450
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The compositions and dosage forms of the invention are clinically useful as methods for increasing the effectiveness, efficiency and safety of biguanides (metformin) and/or sulfonylureas in the prevention and treatment of insulin resistance and diabetes mellitus, alone or in combination, as a nutrient for humans. The carefully chosen active ingredients of the invention are designed in a modular fashion to prevent and rectify adverse events associated with insulin resistance syndrome and diabetes mellitus, and with the clinical use of biguanides (metformin) and/or the sulfonylureas. These modules are: (1) Mitochondrial Metabolic Group, (2) Plasma and Mitochondrial Membrane Integrity Group, (3) Nocturnal Group and, (4) Insulin Alternative Group. When used in concert with a biguanide, a sulfonylurea or with a combination of both, the invention will broaden the clinical usefulness of these drugs. The invention will retard the progression of insulin resistance to type 2 diabetes, and reduce the serious microvascular and macrovascular complications commonly associated with insulin resistance syndrome and diabetes mellitus.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 6 OF 81 USPATFULL
ACCESSION NUMBER: 2003:106745 USPATFULL
TITLE: Use of alpha 1-antichymotrypsin polypeptides, or nucleic acids encoding them, or of a cell which is **expressing** an ACT polypeptide, or a nucleic acid encoding it, for treatment and/or prevention of **diabetes-associated** and/or arterial poorly healing wounds and for identifying pharmacologically active substances

INVENTOR(S): Halle, Jorn-Peter, Penzberg, GERMANY, FEDERAL REPUBLIC OF
Goppelt, Andreas, Munchen, GERMANY, FEDERAL REPUBLIC OF
Hof, Peter, Martinsried, GERMANY, FEDERAL REPUBLIC OF

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003073657	A1	20030417
APPLICATION INFO.:	US 2002-135629	A1	20020430 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	DE 2001-121255	20010430
	US 2001-323348P	20010918 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: CLARK & ELBING LLP, 101 FEDERAL STREET, BOSTON, MA, 02110
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
LINE COUNT: 1977
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to the use of alpha 1-antichymotrypsin (ACT) polypeptides according to SEQ ID No. 1 to SEQ ID No. 4 and/or nucleic acids encoding them, or an antibody or a fragment thereof directed against the polypeptide, or of a cell which is **expressing** the polypeptide or a nucleic acid encoding it, for diagnosis, treatment and/or prevention of **diabetes-associated** and/or arterial wounds which heal poorly and for identifying pharmacologically active substances which exert an influence on the **expression** or function, particularly the activity of ACT.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 7 OF 81 USPATFULL
ACCESSION NUMBER: 2003:100088 USPATFULL
TITLE: Treatment methods based on microcompetition for a limiting GABP complex
INVENTOR(S): Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003069199	A1	20030410
APPLICATION INFO.:	US 2002-219334	A1	20020815 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Hanan Polansky, 3159 S. Winton Rd., Rochester, NY, 14623
NUMBER OF CLAIMS: 26
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 28 Drawing Page(s)
LINE COUNT: 14837
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Microcompetition for GABP between a foreign polynucleotide and a cellular GABP regulated gene is a risk factor associated with chronic disease such as obesity, cancer, atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other autoimmune diseases. The invention uses this novel discovery to present methods for the treatment of these chronic diseases. The methods are based on modifying such microcompetition, or the effect of such microcompetition on the cell. For instance, treatment may modify the cellular copy number of the foreign polynucleotide, change the rate of complex formation between GABP and either the foreign polynucleotide or the cellular GABP

regulated gene, vary the **expression** of the cellular GABP regulated gene, or manipulate the activity of the gene product of the cellular GABP regulated gene. The invention also presents methods for treatment of chronic diseases resulting from other foreign polynucleotide-type disruptions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 8 OF 81 USPATFULL

ACCESSION NUMBER:	2003:99511 USPATFULL
TITLE:	Drug discovery assays based on microcompetition for a limiting GABP complex
INVENTOR(S):	Polansky, Hanan, Rochester, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003068616	A1	20030410
APPLICATION INFO.:	US 2002-223050	A1	20020814 (10)

RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-732360, filed on 7 Dec 2000, PENDING
DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	Hanan Polansky, 3159 S. Winton Rd., Rochester, NY, 14623
NUMBER OF CLAIMS:	55
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	28 Drawing Page(s)
LINE COUNT:	14981

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A recent discovery showed that microcompetition for GABP between a foreign polynucleotide and a cellular GABP regulated gene is a risk factor for some of the major chronic diseases, such as obesity, cancer, atherosclerosis, stroke, osteoarthritis, diabetes, asthma, and other autoimmune diseases. The invention uses this novel discovery to present assays for screening compounds based on their effectiveness in modulating such microcompetition, or the effects of such microcompetition on the cell. The selected compounds can be used in treatment of these chronic diseases. The invention also presents assays for screening compounds that can be used in treatment of chronic diseases resulting from other foreign polynucleotide-type disruptions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 9 OF 81 USPATFULL

ACCESSION NUMBER:	2003:86817 USPATFULL
TITLE:	Immune modulation method using steroid compounds
INVENTOR(S):	Ahlem, Clarence N., San Diego, CA, UNITED STATES Frincke, James M., San Diego, CA, UNITED STATES dos Anjos de Carvalho, Luis Daniel, Paio Pires, PORTUGAL Heggie, William, Palmela, PORTUGAL Prendergast, Patrick T., County Kildare, IRELAND Reading, Christopher L., San Diego, CA, UNITED STATES Thadikonda, Krupakar Paul, Gaithersburg, MD, UNITED STATES Vernon, Russell N., Oak Hills, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003060425	A1	20030327
APPLICATION INFO.:	US 2001-820483	A1	20010329 (9)

RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1999-449184, filed on 24 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-414905, filed on 8 Oct 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-449004, filed

on 24 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 2000-535675, filed on 23 Mar 2000, PENDING Continuation-in-part of Ser. No. US 1999-449042, filed on 24 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 2000-675470, filed on 28 Sep 2000, PENDING Continuation-in-part of Ser. No. US 2000-586673, filed on 1 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-586672, filed on 1 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 1999-461026, filed on 15 Dec 1999, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-109924P	19981124 (60)
	US 1999-140028P	19990616 (60)
	US 1998-109923P	19981124 (60)
	US 1999-126056P	19991019 (60)
	US 1999-124087P	19990311 (60)
	US 1998-110127P	19981127 (60)
	US 1999-161453P	19991025 (60)
	US 1999-145823P	19990727 (60)
	US 1999-137745P	19990603 (60)
	US 1998-112206P	19981215 (60)
	US 2000-257071P	20001220 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOLLIS-EDEN PHARMACEUTICALS, INC., 4435 EASTGATE MALL, SUITE 400, SAN DIEGO, CA, 92121
NUMBER OF CLAIMS: 54
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 14708
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides compositions comprising formula 1 steroids, e.g., 16.alpha.-bromo-3 .beta.-hydroxy-5.alpha.-androstan-17-one hemihydrate and one or more excipients, including compositions that comprise a liquid formulation comprising less than about 3% v/v water. The compositions are useful to make improved pharmaceutical formulations. The invention also provides methods of intermittent dosing of steroid compounds such as analogs of 16.alpha.-bromo-3.beta.-hydroxy-5.alpha.-androstan-17-one and compositions useful in such dosing regimens. The invention further provides compositions and methods to inhibit pathogen replication, ameliorate symptoms associated with immune dysregulation and to modulate immune responses in a subject using the compounds. The invention also provides methods to make and use these immunomodulatory compositions and formulations.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 10 OF 81 USPATFULL
ACCESSION NUMBER: 2003:86302 USPATFULL
TITLE: Nucleic acids, proteins, and antibodies
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Barash, Steven C., Rockville, MD, UNITED STATES
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., Rockville, MD, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003059908	A1	20030327
APPLICATION INFO.:	US 2002-91504	A1	20020307 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-764869, filed on 17 Jan 2001, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-179065P	20000131 (60)
	US 2000-180628P	20000204 (60)
	US 2000-214886P	20000628 (60)
	US 2000-217487P	20000711 (60)
	US 2000-225758P	20000814 (60)
	US 2000-220963P	20000726 (60)
	US 2000-217496P	20000711 (60)
	US 2000-225447P	20000814 (60)
	US 2000-218290P	20000714 (60)
	US 2000-225757P	20000814 (60)
	US 2000-226868P	20000822 (60)
	US 2000-216647P	20000707 (60)
	US 2000-225267P	20000814 (60)
	US 2000-216880P	20000707 (60)
	US 2000-225270P	20000814 (60)
	US 2000-251869P	20001208 (60)
	US 2000-235834P	20000927 (60)
	US 2000-234274P	20000921 (60)
	US 2000-234223P	20000921 (60)
	US 2000-228924P	20000830 (60)
	US 2000-224518P	20000814 (60)
	US 2000-236369P	20000929 (60)
	US 2000-224519P	20000814 (60)
	US 2000-220964P	20000726 (60)
	US 2000-241809P	20001020 (60)
	US 2000-249299P	20001117 (60)
	US 2000-236327P	20000929 (60)
	US 2000-241785P	20001020 (60)
	US 2000-244617P	20001101 (60)
	US 2000-225268P	20000814 (60)
	US 2000-236368P	20000929 (60)
	US 2000-251856P	20001208 (60)
	US 2000-251868P	20001208 (60)
	US 2000-229344P	20000901 (60)
	US 2000-234997P	20000925 (60)
	US 2000-229343P	20000901 (60)
	US 2000-229345P	20000901 (60)
	US 2000-229287P	20000901 (60)
	US 2000-229513P	20000905 (60)
	US 2000-231413P	20000908 (60)
	US 2000-229509P	20000905 (60)
	US 2000-236367P	20000929 (60)
	US 2000-237039P	20001002 (60)
	US 2000-237038P	20001002 (60)
	US 2000-236370P	20000929 (60)
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	US 2000-237040P	20001002 (60)
	US 2000-240960P	20001020 (60)
	US 2000-239935P	20001013 (60)
	US 2000-239937P	20001013 (60)
	US 2000-241787P	20001020 (60)
	US 2000-246474P	20001108 (60)
	US 2000-246532P	20001108 (60)
	US 2000-249216P	20001117 (60)
	US 2000-249210P	20001117 (60)
	US 2000-226681P	20000822 (60)
	US 2000-225759P	20000814 (60)
	US 2000-225213P	20000814 (60)
	US 2000-227182P	20000822 (60)
	US 2000-225214P	20000814 (60)
	US 2000-235836P	20000927 (60)
	US 2000-230438P	20000906 (60)

US 2000-215135P 20000630 (60)
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US 2000-249208P 20001117 (60)
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US 2000-249207P 20001117 (60)
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US 2000-251988P 20001205 (60)
US 2000-251030P 20001205 (60)
US 2000-251479P 20001206 (60)
US 2000-256719P 20001205 (60)
US 2000-250160P 20001201 (60)
US 2000-251989P 20001208 (60)
US 2000-250391P 20001201 (60)
US 2000-254097P 20001211 (60)
US 2000-231968P 20000912 (60)
US 2000-226279P 20000818 (60)
US 2000-186350P 20000302 (60)
US 2000-184664P 20000224 (60)

	US 2000-189874P	20000316 (60)
	US 2000-198123P	20000418 (60)
	US 2000-227009P	20000823 (60)
	US 2000-235484P	20000926 (60)
	US 2000-190076P	20000317 (60)
	US 2000-209467P	20000607 (60)
	US 2000-205515P	20000519 (60)
	US 2001-259678P	20010105 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
LINE COUNT: 28555
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel cardiovascular system related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "cardiovascular system antigens," and the use of such cardiovascular system antigens for detecting disorders of the cardiovascular system, particularly the presence of cancer of cardiovascular system tissues and cancer metastases. More specifically, isolated cardiovascular system associated nucleic acid molecules are provided encoding novel cardiovascular system associated polypeptides. Novel cardiovascular system polypeptides and antibodies that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human cardiovascular system associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the cardiovascular system, including cancer of cardiovascular system tissues, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 11 OF 81 USPATFULL
ACCESSION NUMBER: 2003:37137 USPATFULL
TITLE: Compositions and methods for regulating endogenous inhibitor of ATP synthase, including treatment for diabetes
INVENTOR(S): Anderson, Christen Marie, Encinitas, CA, UNITED STATES
Clevenger, William, Oceanside, CA, UNITED STATES
PATENT ASSIGNEE(S): MitoKor, San Diego, CA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003026781	A1	20030206
APPLICATION INFO.:	US 2002-83815	A1	20020227 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-796076, filed on 27 Feb 2001, PENDING Continuation-in-part of Ser. No. US 2000-709189, filed on 10 Nov 2000, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-164622P	19991110 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092
NUMBER OF CLAIMS: 85

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 5927
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides compositions and methods for altering mitochondrial ATP metabolism, including compositions having fusion proteins comprising IF1 polypeptide-derived sequences, as well as binding and functional assays exploiting IF1 interactions with ATP synthase. Also disclosed are methods for identifying an agent capable of reducing mitochondrial ATP hydrolysis and/or increasing mitochondrial ATP synthesis, including pharmaceutical compositions identified by such methods. The invention also provides methods for treating diabetes, and in particular, type 2 DM, using an agent identified according to the disclosed methods.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 12 OF 81 USPATFULL
ACCESSION NUMBER: 2003:18018 USPATFULL
TITLE: Composition, synthesis and therapeutic applications of polyamines
INVENTOR(S): Murphy, Michael A., La Jolla, CA, UNITED STATES
MaLachowski, Mitchell R., San Diego, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003013772	A1	20030116
APPLICATION INFO.:	US 2001-17235	A1	20011218 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-486310, filed on 23 Feb 2000, PENDING A 371 of International Ser. No. WO 1998-US17301, filed on 21 Aug 1998, UNKNOWN A 371 of International Ser. No. US 1997-915660, filed on 21 Aug 1997, GRANTED, Pat. No. US 5906996
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: CHARMASSON & BUCHACA, 1545 HOTEL CIRCLE SOUTH, SUITE 150, SAN DIEGO, CA, 92108-3412
NUMBER OF CLAIMS: 74
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 3034
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to the preparation of novel polyamines, such as derivatives of 1,3-bis-[(2'-aminoethyl)-amino]propane (2,3,2-tetramine) and 1,4,8,11-tetraazacyclotetradecane (cyclam), which can be used to treat mitochondrial and degenerative diseases.

Accordingly, in one aspect the invention is directed to compounds of the formula: ##STR1##

wherein

R.sub.1, R.sub.2, R.sub.3, R.sub.4, R.sub.5 and R.sub.6 may be the same or different and are hydrogen, alkyl, aryl, cycloalkyl, amino acid, glutathione, urate, ascorbate, estrogen, dehydroepiandrosterone, redox stabilizing substituents, a quinone, glutamate, succinate, --(CH.sub.2).sub.n[XCH.sub.2).sub.n]NH.sub.2-- wherein n=3-6 and X=nitrogen, sulfur, phosporous or carbon, or heterocycle wherein R.sub.1 to R.sub.6 taken together are --(CH.sub.2XCH.sub.2).sub.n-- wherein n=3-6 and X=nitrogen, sulfur, phosporous or carbon.

M, n, and p may be the same or different and are bridging groups of variable length from 3-12 carbons.

X.sub.1 and X.sub.2 may be the same or different and are nitrogen,

sulfur, phosporous or carbon.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 13 OF 81 USPATFULL
ACCESSION NUMBER: 2003:3447 USPATFULL
TITLE: MRP8/MRP14 heterodimer, or its individual components in combination, for treating and/or preventing skin diseases, wounds and/or wound-healing disturbances, having a reduced quantity of MRP8/MRP14 heterodimers
INVENTOR(S): Halle, Jorn-Peter, Penzberg, GERMANY, FEDERAL REPUBLIC OF
Goppelt, Andreas, Munchen, GERMANY, FEDERAL REPUBLIC OF

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003003482	A1	20030102	
APPLICATION INFO.:	US 2002-134841	A1	20020429	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	DE 2001-121254	20010430
	US 2001-322925P	20010917 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: CLARK & ELBING LLP, 101 FEDERAL STREET, BOSTON, MA, 02110
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 2013
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to the use of an MRP8/MRP14 heterodimer, or of its individual components in combination, of at least one nucleic acid encoding the entire heterodimer or its individual components in combination, or of a cell which is **expressing** the entire heterodimer, or its individual components in combination, for treating and/or preventing skin diseases, wounds, and/or wound-healing disturbances having a reduced quantity of MRP8/MRP14 heterodimers, in particular **diabetes-associated** wounds, and to methods for identifying pharmacologically active substances which exert an influence on the function or **expression** of MRP8/MRP14 heterodimers.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 14 OF 81 USPATFULL
ACCESSION NUMBER: 2003:130002 USPATFULL
TITLE: Peptide epitopes recognized by disease promoting CD4+ T lymphocytes
INVENTOR(S): Peakman, Mark, London, UNITED KINGDOM
Chicz, Roman M., Belmont, MA, United States
PATENT ASSIGNEE(S): Zycos, Inc., Cambridge, MA, United States (U.S. corporation)
King's College London, London, UNITED KINGDOM (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6562943	B1	20030513	
APPLICATION INFO.:	US 2000-552802		20000420	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-130355P	19990421 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Nolan, Patrick J.
ASSISTANT EXAMINER: DeCloux, Amy
LEGAL REPRESENTATIVE: Fish & Richardson P.C.
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT: 2359
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides methods for identifying peptide epitopes that activate CD4+ T cells involved in the pathogenesis of diseases, e.g., autoimmune diseases, susceptibility to which is determined by **expression** of particular class II MHC genes. The invention includes peptides derived from the IA-2 polypeptide by such a method, altered peptide ligands, and methods of therapy involving the use of altered peptide ligands.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 15 OF 81 USPATFULL
ACCESSION NUMBER: 2003:123328 USPATFULL
TITLE: Formulated composition
INVENTOR(S): Oeswein, James Q., Moss Beach, CA, United States
Smikahl, John R., Foster City, CA, United States
Wang, Sharon X., San Mateo, CA, United States
Yeung, Douglas A., Fremont, CA, United States
PATENT ASSIGNEE(S): Genentech, Inc., South San Francisco, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6559122	B1	20030506	
APPLICATION INFO.:	US 2000-539310		20000330	(9)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 1999-128392P	19990408	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Low, Christopher S. F.
ASSISTANT EXAMINER: Mohamed, Abdel A.
LEGAL REPRESENTATIVE: Hasak, Janet E.
NUMBER OF CLAIMS: 32
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 1577
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A composition is disclosed that comprises a mixture of polypeptides of opposite charge and an excipient selected from the group consisting of arginine, lysine, glutamic acid, sodium dodecyl sulfate, beta-hydroxy cyclodextrin, and beta-cyclodextrin sulfobutyl ether.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 16 OF 81 USPATFULL
ACCESSION NUMBER: 2003:123105 USPATFULL
TITLE: Methods for manipulating upper gastrointestinal transit, blood flow, and satiety, and for treating visceral hyperalgesia
INVENTOR(S): Lin, Henry C., Manhattan Beach, CA, United States
PATENT ASSIGNEE(S): Cedars-Sinai Medical Center, Los Angeles, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6558708	B1	20030506
APPLICATION INFO.:	US 2000-546119		20000410 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1999-420046, filed on 18 Oct 1999 Continuation-in-part of Ser. No. US 1999-359583, filed on 22 Jul 1999, now abandoned Continuation of Ser. No. US 1997-832307, filed on 3 Apr 1997, now patented, Pat. No. US 5977175, issued on 2 Nov 1999 Continuation of Ser. No. US 1995-442843, filed on 17 May 1995, now abandoned

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Page, Thurman K.
ASSISTANT EXAMINER: Tran, S.
LEGAL REPRESENTATIVE: Sidley Austin Brown & Wood LLP
NUMBER OF CLAIMS: 13
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Figure(s); 6 Drawing Page(s)
LINE COUNT: 3377
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed are a method of manipulating the rate of upper gastrointestinal transit of a substance in a mammal. Also disclosed are methods of manipulating satiety and post-prandial visceral blood flow. A method of treating visceral pain or visceral hypersensitivity in a human subject is also described. A method for prolonging the residence time of an orally or enterally administered substance by promoting its dissolution, bioavailability and/or absorption in the small intestine is also described. These methods are related to a method of transmitting to and replicating at a second location in the central nervous system a serotonergic neural signal originating at a first location in the proximal or distal gut of a mammal and/or a method of transmitting to and replicating at a second location in the upper gastrointestinal tract a serotonergic neural signal originating at a first location in the proximal or distal gut.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 17 OF 81 USPATFULL
ACCESSION NUMBER: 2003:53520 USPATFULL
TITLE: Prevention and treatment of retinal ischemia and edema
INVENTOR(S): Adamis, Anthony P., Jamaica Plain, MA, United States
PATENT ASSIGNEE(S): The Children's Medical Center Corporation, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6524581	B1	20030225
APPLICATION INFO.:	US 1999-474523		19991229 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1999-248752, filed on 12 Feb 1999

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-114221P	19981230 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Wang, Andrew
ASSISTANT EXAMINER: Schultz, James S.
LEGAL REPRESENTATIVE: Hamilton, Brook, Smith & Reynolds, P.C.
NUMBER OF CLAIMS: 26
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 38 Drawing Figure(s); 16 Drawing Page(s)
LINE COUNT: 2386
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to methods of treating retinopathy,

retinal ischemia and/or retinal edema comprising administering an integrin or integrin subunit antagonist, leukocyte adhesion-inducing cytokine antagonist or growth factor antagonist, a selectin antagonist or adhesion molecule antagonist.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 18 OF 81 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 2
ACCESSION NUMBER: 2003:308540 CAPLUS
TITLE: Association of an IL-1A 3'UTR polymorphism with end-stage renal disease and **IL-1** .alpha. **expression**
AUTHOR(S): Bensen, Jeannette T.; Langefeld, Carl D.; Li, Liwu; McCall, Charles E.; Cousart, Susan L.; Dryman, Bonnie N.; Freedman, Barry I.; Bowden, Donald W.
CORPORATE SOURCE: The Center for Human Genomics and Department of Public Health Sciences, Department of Internal Medicine, and Department of Biochemistry, Wake Forest University School of Medicine, Winston-Salem, NC, USA
SOURCE: Kidney International (2003), 63(4), 1211-1219
CODEN: KDYIA5; ISSN: 0085-2538
PUBLISHER: Blackwell Publishing, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Background. We evaluated polymorphisms in the interleukin-1 alpha 3'-untranslated region (IL-1A 3'[UTR]) for assocn. with type 2 **diabetes-assocd**. (DM) and nondiabetic-assocd. (non-DM) end-stage renal disease (ESRD) in two ethnic groups. IL-1A 3'UTR polymorphisms were identified by alignment of overlapping human **expressed** sequence tags (ESTs). Sequence ambiguities were exptl. confirmed and variants genotyped to test for assocn. with ESRD in 75 unrelated Caucasians with DM ESRD, 95 unrelated Caucasian controls and, in a parallel study, 92 unrelated African Americans with type 2 DM ESRD, 95 unrelated African Americans with non-DM ESRD, and 86 unrelated African American controls. IL-1A 3' UTR genotype and lipopolysaccharide (LPS)-stimulated **IL-1**.alpha. protein levels were measured in healthy Caucasians (N = 112) and African Americans (N = 101) to evaluate assocn. between genotype and protein level. A polymorphism in the 3' UTR of the human IL-1A gene was assocd. with ESRD and **IL-1**.alpha. protein **expression**. The polymorphism consists of two single nucleotide polymorphisms (SNPs) and an insertion/deletion generating four different haplotypes: TN7TTCAA, AN7TTCAA, TN7TTCAG and an allele deleted for four internal bases, TN7(delTTCA)A. The 4 bp deletion allele, TN7(delTTCA)A, was significantly less common among Caucasian DM ESRD and African American non-DM ESRD patients (recessive model; P = 0.0364 and P = 0.0293, resp.). In vitro, this polymorphism is assocd. with the amt. of **IL-1**.alpha. protein synthesized in LPS-stimulated lymphocytes from healthy subjects (P = 0.0013, additive model), with the TN7(delTTCA)A haplotype assocd. with higher levels of stimulated **IL-1**.alpha.. Conclusion. The assocn. of the TN7(delTTCA)A haplotype with higher levels of **IL-1** .alpha. **expression** and reduced risk for ESRD is consistent with involvement of cytokines in risk for **developing** nephropathy.
REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 19 OF 81 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:153973 CAPLUS
DOCUMENT NUMBER: 138:236749
TITLE: Enterovirus infection may induce humoral immune response reacting with islet cell autoantigens in humans
AUTHOR(S): Harkonen, Taina; Paananen, Anja; Lankinen, Hilkka; Hovi, Tapani; Vaarala, Outi; Roivainen, Merja
CORPORATE SOURCE: Enterovirus Laboratory, National Public Health

Institute (KTL), Helsinki, Finland
SOURCE: Journal of Medical Virology (2003), 69(3), 426-440
CODEN: JMVIDB; ISSN: 0146-6615
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Mol. mimicry is one of the mechanisms by which enterovirus infections have been postulated to have a role in the pathogenesis of type 1 diabetes. Immunogenic epitopes in enterovirus capsid protein VP1 and procapsid protein VP0 have sequence similarities with **diabetes-assocd.** epitopes in tyrosine phosphatase IA-2/IAR and **heat shock** protein 60. In the present study, documented enterovirus infection was shown to induce humoral responses, that in 7% and 1% of patients cross-reacted with the known **diabetes-assocd.** epitopes in tyrosine phosphatase IAR and **heat shock** protein 60, resp. In contrast, none of the children vaccinated against poliomyelitis had antibodies to the **diabetes-assocd.** epitope of tyrosine phosphatases IA2/IAR. The antibody response studied in serum samples from six patients with coxsackievirus A9 infection was mainly targeted to capsid protein VP1. Coxsackievirus A9 infection induced antibodies cross-reacted with one epitope in **heat shock** protein 60, but not with epitopes derived from other autoantigens. Most diabetic children had high levels of antibodies to both coxsackievirus and poliovirus derived VP1 peptides but the pattern of reactivity did not differ from that seen in healthy children. The reactivity of linear epitopes derived from autoantigens was low in general and assocd. with the presence of multiple autoantibodies in the patients. Some linear auto-epitopes derived from tyrosine phosphatase IA-2, glutamic acid decarboxylase 65, preproinsulin, and **heat shock** protein 60 were recognized by sera from diabetic patients, but not by sera from healthy children. In conclusion, enteroviruses may induce immune responses that react with islet cell autoantigens, which is a concern when a putative inactivated enterovirus vaccine is considered.

REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L25 ANSWER 20 OF 81 USPATFULL DUPLICATE 3
ACCESSION NUMBER: 2002:227678 USPATFULL
TITLE: Therapeutic agent for treatment of diabetes
INVENTOR(S): Kishino, Michiko, Suita-shi, JAPAN
Nakayama, Chikao, Sanda-shi, JAPAN
Taiji, Mutsuo, Takatsuki-shi, JAPAN
Ichihara, Junji, Takatsuki-shi, JAPAN
Noguchi, Hiroshi, Kawanishi-shi, JAPAN
PATENT ASSIGNEE(S): Sumitomo Pharmaceuticals Co., Limited (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002122829	A1	20020905
	US 6472366	B2	20021029
APPLICATION INFO.:	US 2002-55380	A1	20020125 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 1999-355098, filed on 23 Jul 1999, PENDING A 371 of International Ser. No. WO 1998-JP157, filed on 19 Jan 1998, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	JP 1997-26111	19970123
	JP 1997-102478	19970404
	JP 1997-102479	19970404

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BIRCH STEWART KOLASCH & BIRCH, PO BOX 747, FALLS CHURCH, VA, 22040-0747

NUMBER OF CLAIMS: 4
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 924
AB The present invention provides a therapeutic agent for treatment of diabetes and hyperlipemia, especially a therapeutic agent for treatment of type II diabetes mellitus, which comprises as the active ingredient a neurotrophic factor such as BDNF (brain-derived neurotrophic factor), ligands of trkB or trkC receptors, NGF, NT-3, NT-4/5, CNTF, GDNF, HGF, etc. Different from conventional oral hypoglycemic agents being mainly used in the treatment of type II diabetes mellitus, the agent of the present invention exhibit blood lipid regulating effects and body fat accumulation regulating effects, in addition to the blood glucose regulating effects. Thus, the agent of the present invention are novel, and can reduce the risk factors in diabetes accompanied by hyperlipemia or obesity, without using any other agent.

L25 ANSWER 21 OF 81 USPATFULL DUPLICATE 4
ACCESSION NUMBER: 2002:198274 USPATFULL
TITLE: Novel methods and compositions to upregulate, redirect or limit immune responses to peptides, proteins and other bioactive compounds and vectors **expressing** the same
INVENTOR(S): Bot, Adrian, San Diego, CA, UNITED STATES
Dellamary, Luis, San Marcos, CA, UNITED STATES
Smith, Dan J., San Diego, CA, UNITED STATES
Woods, Catherine M., La Jolla, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002106368	A1	20020808	
APPLICATION INFO.:	US 2001-919477	A1	20010730	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-221544P	20000728 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KNOBBE MARTENS OLSON & BEAR LLP, 620 NEWPORT CENTER DRIVE, SIXTEENTH FLOOR, NEWPORT BEACH, CA, 92660
NUMBER OF CLAIMS: 80
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 31 Drawing Page(s)
LINE COUNT: 2513
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel compositions are disclosed which can induce or enhance an immune response against foreign or self antigens (microbial or parasitic) or modulate (that can lead to suppression) the activity of pathogenic cells in inflammatory or autoimmune diseases. Compositions and methods are taught in how to limit the generation of an immune response against formulated peptides and proteins with application in antibody therapy or hormone replacement therapy. Methods of suppressing autoimmunity are also disclosed which use ligands for cellular receptors **expressed** on cells of the innate immune system and more specifically for down-regulation of autoimmune processes by either deletion or induction of anergy at the level of autoreactive T cells or by triggering active-suppressor T cells that down-regulate the activity of pathogenic cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 22 OF 81 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 5
ACCESSION NUMBER: 2002:927718 CAPLUS
DOCUMENT NUMBER: 138:12503

TITLE: Mammalian **diabetes-mediating** proteins identification for diagnosis and therapy
INVENTOR(S): Larsen, Peter Mose; Fey, Stephen J.; Karlsen, Allan E.; Sparre, Thomas; Nerup, Jorn
PATENT ASSIGNEE(S): Syddansk Universitet, Den.
SOURCE: PCT Int. Appl., 128 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002097441	A2	20021205	WO 2002-DK368	20020529

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: DK 2001-852 A 20010529
DK 2002-446 A 20020322

AB Provided are mammalian secreted and non-secreted **diabetes mediating** proteins, including protective and deleterious **diabetes-mediating** proteins, as well as polynucleotides encoding same, drug screening methods for identifying a test compd. capable of altering the **expression** of a **diabetes-mediating** protein, and methods of preventing or ameliorating diabetes by administering a compd. capable of altering the **expression** of a **diabetes-mediating** protein. The proteins were identified by monitoring **IL-1**.beta. induced protein changes in diabetes prone mammalian islets of Langerhans using two-dimensional gel electrophoresis. Protein spots that significantly changed **expression** levels after exposure to **IL-1**.beta. were cut out of the gels and subjected to MALDI mass spectrometry. Eighty-two significantly changed protein spots were detected. Pos. identification was obtained for a total of 45 different proteins from 51 of the 82 spots.

L25 ANSWER 23 OF 81 USPATFULL
ACCESSION NUMBER: 2002:329468 USPATFULL
TITLE: Antigen specific recombinant MHC class II molecules and methods of use
INVENTOR(S): Liu, Chih-Pin, Cerritos, CA, UNITED STATES
Lin, Wei-Jen, Cerritos, CA, UNITED STATES
PATENT ASSIGNEE(S): City of Hope, Duarte, CA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002187147	A1	20021212	
APPLICATION INFO.:	US 2002-74257	A1	20020214	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-268714P	20010215 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ROTHWELL, FIGG, ERNST & MANBECK, P.C., 1425 K STREET, N.W., SUITE 800, WASHINGTON, DC, 20005

NUMBER OF CLAIMS: 48
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 26 Drawing Page(s)
LINE COUNT: 1642
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to specific, multivalent, soluble recombinant I-Ag7/peptide complexes. These complexes are stable and are recognized by the TCR of T cells specific for a preselected antigen, to which they bind with low affinity. The inventive complexes may be used, for example, in the identification and study of autoreactive T cells and in the diagnosis, treatment and/or prevention of autoimmune or other diseases, including diabetes or pre-diabetic conditions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 24 OF 81 USPATFULL
ACCESSION NUMBER: 2002:322564 USPATFULL
TITLE: Method for transdifferentiation of non pancreatic stem cells to the pancreatic differentiation pathway
INVENTOR(S): Ramiya, Vijayakumar, Gainesville, FL, UNITED STATES
Clark, Amy, Gainesville, FL, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002182728	A1	20021205
APPLICATION INFO.:	US 2002-113118	A1	20020329 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-279922P	20010329 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SWANSON & BRATSCHUN L.L.C., 1745 SHEA CENTER DRIVE, SUITE 330, HIGHLANDS RANCH, CO, 80129
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 775
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The subject invention comprises culture methods for transdifferentiation of non-pancreatic stem cells to the pancreatic differentiation pathway. It also concerns the endocrine hormones that can be produced by such cultures, and the use of the transdifferentiated cells in the treatment of pancreatic diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 25 OF 81 USPATFULL
ACCESSION NUMBER: 2002:272775 USPATFULL
TITLE: Methods of treatment of type 2 diabetes
INVENTOR(S): Polonsky, Kenneth S., Chicago, IL, UNITED STATES
Horikawa, Yukio, Kobe City, JAPAN
Oda, Naohisa, Nagoya-shi, JAPAN
Cox, Nancy J., Inverness, IL, UNITED STATES
Otani, Kenichi, Chicago, IL, UNITED STATES
Hanis, Craig L., Houston, TX, UNITED STATES
Bell, Graeme I., Chicago, IL, UNITED STATES
Sreenan, Seamus Kevin, Dublin 3, IRELAND
Zhou, Yun-Ping, Pleasanton, CA, UNITED STATES
PATENT ASSIGNEE(S): Board of Regents, The University of Texas System (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002150896	A1	20021017

APPLICATION INFO.: US 2001-768877 A1 20010123 (9)
RELATED APPLN. INFO.: Division of Ser. No. US 1999-422869, filed on 21 Oct 1999, GRANTED, Pat. No. US 6235481

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-105052P	19981021 (60)
	US 1999-134175P	19990513 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Gina N. Shishima, FULBRIGHT & JAWORSKI L.L.P., Suite 2400, 600 Congress Avenue, Austin, TX, 78701
NUMBER OF CLAIMS: 48
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 26 Drawing Page(s)
LINE COUNT: 8520
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates generally to the field of diabetes. More particularly, it concerns the identification of genes responsible for NIDDM1 for use in diagnostic and therapeutic applications. The present invention demonstrates that the NIDDM1 locus is, in fact, the calpain 10 gene. The invention further relates to the discovery that analysis of mutations in calpain genes and gene products can be diagnostic for type 2 diabetes. The invention also contemplates methods of treating diabetes in view of the fact that calpain mutations can cause diabetes. Further, the invention relates to novel polynucleotides of the NIDDM1 locus and polypeptides encoded by such polynucleotides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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